RADIOLOGY

A MONTHLY JOURNAL DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES

Vol. 53 AUGUST, 1949 No. 2

CONTENTS

RADIOLOGY

A MONTHLY PUBLICATION DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES

PUBLISHED BY THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

GENERAL INFORMATION

RADIOLOGY is entered as second class matter at Syracuse, New York, and Easton, Penna., under the Act of August 24, 1912, and accepted November 24, 1934. RADIOLOGY is published by the Radiological Society of North America as its official Journal. Subscription rate $8.00 per annum. Canadian postage, $1.00 additional. Foreign postage, $2.00 additional. Single copies $1.00 each. All correspondence relative to business matters connected with the Radiological Society of North America and RADIOLOGY, or remittance for non-member subscriptions, should be made payable to the Radiological Society of North America and should be addressed to the BUSINESS MANAGER, DONALD S. CHILDS, M.D., 713 E. GENESEE STREET, SYRACUSE 2, NEW YORK. In requesting change of address, both the old and the new address should be given.

Dues to the Radiological Society of North America include subscription to RADIOLOGY and should be paid to DONALD S. CHILDS, M.D., SECRETARY-TREASURER, 713 E. GENESEE STREET, SYRACUSE 2, N. Y.

The rate for "want" advertisements for insertion in the Classified Section is 8 cents per word, minimum charge $2.00. Remittance should accompany order. Rates for display advertisements will be furnished upon request.

Inquiries regarding the program for the Annual Meeting of the Society for the current year should be sent to the President.

RADIOLOGY is published under the supervision of the Publication Committee of the Radiological Society of North America, who reserve the right to reject any material submitted for publication, including advertisements. No responsibility is accepted by the Committee or the Editor for the opinions expressed by the contributors, but the right is reserved to introduce such changes as may be necessary to make the contributions conform to the editorial standards of RADIOLOGY. Correspondence relating to publication of papers should be addressed to the Editor, HOWARD P. DOUB, M.D., HENRY FORD HOSPITAL, DETROIT 2, MICHIGAN.

Original articles will be accepted only with the understanding that they are contributed solely to RADIOLOGY. Articles in foreign languages will be translated if they are acceptable. Manuscripts should be typewritten, double-spaced, with wide margins, on good paper, and the original, not a carbon copy, should be submitted. The author's full address should appear on the manuscript. It is advisable that a copy be retained for reference as manuscripts will not be returned.

Illustrations and tables should be kept within reasonable bounds, as the number which can be published without cost to the author is strictly limited. For excess figures and for illustrations in color, estimates will be furnished by the Editor. Photographic prints should be clear and distinct and on glossy paper. Drawings and charts should be in India ink on white or on blue-lined coordinate paper. Blueprints will not reproduce satisfactorily. All photographs and drawings should be numbered, the top should be indicated, and each should be accompanied by a legend with a corresponding number. Authors are requested to indicate on prints made from photomicrographs the different types of cells to which attention is directed, by drawing lines in India ink and writing in the margin. The lines will be reproduced, and the words will be set in type. Attention should be called to points which should be brought out in completed illustrations, by tracings and suitable texts. These instructions should be concise and clear.

As a convenience to contributors to RADIOLOGY who are unable to supply prints for their manuscripts, the Editor can arrange for intermediate prints from roentgenograms.

The Society will furnish fifty reprints to authors, for which arrangements must be made with the Editor.

RADIOLOGY

A MONTHLY JOURNAL DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES

PUBLISHED BY THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

Vol. 53 AUGUST 1949 No. 2

Spontaneous Pneumothorax: A Study of 105 Cases[1]

LOUIS A. ROTTENBERG, M.D., and ROSS GOLDEN, M.D.

New York, N. Y.

SPONTANEOUS pneumothorax is a condition in which air is present outside the lung in the thoracic cavity without obvious portal of entry through the chest wall.

The term "pneumothorax" was initially employed by Itard in 1803, when he reported 5 cases, the diagnosis of which was made at autopsy. McDowell in 1856 described the first clear-cut case of spontaneous pneumothorax in an apparently healthy individual. Kjaergaard contributed his monumental work on this subject in 1932. Several hundred cases have since been reported in the literature (exclusive of figures from the military medical services).

Many adjectives have been used to describe the pneumothorax. "Benign," "true," "real," "idiopathic," "simple," and "spontaneous pneumothorax in apparently healthy individuals" are but a few of the terms. Regardless of terminology, the benign nature of the disease has been stressed by most of the recent investigators.

In many medical textbooks the commonest cause of spontaneous pneumothorax is stated to be pulmonary tuberculosis. The purpose of this paper is to present a study of 105 consecutive cases of spontaneous pneumothorax observed at the Columbia-Presbyterian Medical Center, from 1930 to 1947, inclusive, to determine its possible relationship to pulmonary tuberculosis.

PATHOGENESIS

The three most important etiological agents in the pathogenesis of spontaneous pneumothorax have been considered to be (1) pulmonary tuberculosis, (2) congenital vesicles, and (3) emphysema.

(*1*) *Pulmonary Tuberculosis:* Pulmonary tuberculosis has not infrequently been associated with spontaneous pneumothorax. This is apparently based on certain reports in the literature. For example, Fishberg (1932) in his textbook *Pulmonary Tuberculosis*, remarked that "the consensus of opinion is that the vast majority [of cases of spontaneous pneumothorax] are caused by a tuberculous lesion in the lung or pleura; it is maintained that at least 90 per cent originate thus. The rent in the pleura may be due to softening and consequent perforation of a subpleural tubercle, or to a tear in that membrane caused by tugging of an adhesion.... In the writer's opinion probably about 20 per cent of cases of really spontaneous pneumothoraces are not due to tuberculosis."

[1] From the Department of Radiology of the College of Physicians and Surgeons of Columbia University and the Radiological Service of the Presbyterian Hospital, New York. Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

TABLE I: REPORTED CASES OF SIMPLE SPONTANEOUS PNEUMOTHORAX

Author	No. of Cases	Years	Source
Kjaergaard	51	1909–29	Denmark
Wood	71	–1931	Mayo Clinic
Perry	85	1924–39	London Hospital
Blackford	15	–1939	University of Virginia
Sokoloff	22	–1939	Coal miners
Norris	25	1935–40	Rochester, New York
Griffin	10	1933–41	University of Kentucky
Ornstein and Lercher	58	1922–42	Private practice, New York
Niehaus	24	1927–43	University of Iowa and San Diego
Babington	13	–1944	California
Myerson	36	1934–43	Boston City Hospital
Hyde and Hyde	63	–1947	Los Angeles and Bellevue Hospital, New York
Rottenberg and Golden	97	1930–47	Presbyterian Hospital, New York
TOTAL	570		

Rubin stated, in his textbook *Diseases of the Chest*, that "it was the occasional 'providential' outcome of a spontaneous pneumothorax in the tuberculous that led to the adoption of the procedure as a therapeutic measure."

Kjaergaard (1932) reported 51 cases of spontaneous pneumothorax, and was able to follow 49 of these patients from two to fifteen years. In only one of his cases did pulmonary tuberculosis develop subsequent to the pneumothorax. This patient was exposed to tuberculosis in her home after hospitalization for the pneumothorax, and, as the site of the tuberculosis was in the contralateral lung, he considered the two lesions unrelated.

Perry (1939) reported 85 cases of benign spontaneous pneumothorax from the London Hospital during the period 1924 to 1937, and was able to trace 67 of these from one to five years. He did not find a single case of pulmonary tuberculosis developing in this group. During this fourteen-year period at the same hospital, 16 cases of pneumothorax were observed which were associated with chronic pulmonary tuberculosis. In this latter group, 9 patients died within one month, a mortality rate of 56 per cent. Perry surveyed the literature, which included about 250 recorded cases of benign spontaneous pneumothorax, and was able to find only 6 cases in which chronic pulmonary tuberculosis later developed. He stated that no case had been reported in which a minimal subpleural tubercle was found at autopsy which was responsible for the pneumothorax. Perry thus concluded that the incidence of tuberculosis following spontaneous pneumothorax would be 2 per cent, which is about the same as the percentage for pulmonary tuberculosis in the general London community.

Ornstein and Lercher (1942) reported 58 cases of spontaneous pneumothorax, in 3 of which pulmonary tuberculosis developed in about two years. All of these 3 patients disclosed roentgen evidence of pleural adhesions at the time of the pneumothorax. This subject will be discussed later.

Myerson (1948) reported 100 consecutive cases of spontaneous pneumothorax admitted to the Boston City Hospital between 1934 and 1943. Sixty-four of this group had underlying pulmonary disease, 38 of whom had tuberculosis. Thirty-six cases occurred in apparently healthy individuals. Therefore, in a ten-year period in a large city hospital, spontaneous pneumothorax occurred about as frequently in apparently healthy individuals as in patients with pulmonary tuberculosis.

Fischer claimed that the rupture of a scar-tissue vesicle was the etiological factor. The vesicle was supposedly produced by localized atrophy and inflation of the lung tissue. A valve mechanism theoretically resulted from the localized inflammatory process, with subsequent scar formation and bronchiolar constriction. Hayashi (1915) examined these vesicles microscopically and found a valve-like structure at their base. The valve consisted of atrophic lung tissue which permitted air to pass freely into the vesicle but prevented escape of the air. The vesicle could increase in size gradually, thin out, and finally rupture. Neither

Fischer nor Hayashi offered definite suggestions as to the origin of this scar tissue, and they were unable to demonstrate positive evidence for tuberculosis. Yet, they considered an old, limited tuberculous infection as the most probable cause.

A French radiologist visiting the Presbyterian Hospital in 1947 related an interesting tale to one of us (L. A. R.) relative to the prevalent association of pneumothorax and pulmonary tuberculosis. During the Nazi occupation of France, a demand was made for slave labor. To prevent the wholesale seizure of young Frenchmen, a group of local physicians produced artificial pneumothorax in many of these young adults, using a fine needle; placed the "patients" in sanatoria, and made chest films. The German medical officers were satisfied that spontaneous pneumothorax was present which was complicating pulmonary tuberculosis. The air in the thoracic cavity was renewed several times. Months later the Germans became suspicious. To continue the delusion, normal saline was injected into a knee or into any peripheral joint; a plaster cast was applied, a film was made, and tuberculosis of the joint was thus suggested. This medical "passive resistance" saved a number of young Frenchmen from labor within the enemy camp.

(2) *Congenital Vesicles:* Schmincke (1928) reported a case of bilateral spontaneous pneumothorax in which an autopsy was performed. The lungs and pleura showed no signs of inflammatory changes, but many emphysematous bullae in various stages of development were present in both lungs. Schmincke believed that a developmental alteration in the pulmonary tissue was responsible for these bullae, with a persistence of a peripheral zone of embryonal tissue which did not differentiate into alveoli. These areas could conceivably be changed into cystic areas during respiration, and if a check-valve mechanism should develop, the cysts would enlarge and rupture.

Kjaergaard (1933) reported two autopsies in which the pneumothorax resulted from such a condition, thus confirming Schmincke's original observation. In the literature there are three reports on the familial occurrence of spontaneous pneumothorax with no history of tuberculosis. Two of our cases occurred in brothers, one of whom has been followed for over nine years, and appears in excellent health.

Folke (1935) reviewed the literature on persistent pneumothorax in infants, unassociated with trauma, and collected 20 cases. The patients were all under four months of age, and 9 died. Caffey states he has never seen a case of simple spontaneous pneumothorax at the Babies Hospital, New York.

(3) *Emphysema:* Generalized emphysema had been considered a factor in the etiology. Perry reported that 18 of his 85 patients with spontaneous pneumothorax suffered from this disease. Kjaergaard suggested that generalized emphysema was infrequent in association with spontaneous pneumothorax because the emphysematous bullae are rarely valve-vesicles, and usually communicate with the underlying lung tissue. In postmortem lungs inflated *in situ*, West (1884) found that a rupture of the pleura could not be produced until the pressure was above 200 mm. of mercury. Such a great intrapulmonary pressure is very unusual, and a normal pleura would not rupture through coughing or muscular exertion. West concluded that a pathological condition must exist at the site of the pleural rupture.

Localized emphysema in the form of subpleural bullae appears to be the usually accepted explanation at present for the pathogenesis of simple spontaneous pneumothorax. Miller states that the bullae are produced by the rupture of the elastic fibers of the subpleural alveoli into the areolar layer of the visceral pleura (every case he studied was associated with emphysema). The visceral pleura is elevated and separated from the underlying alveoli. Air forced from the bases of the lungs into the apices of the upper lobes distends and finally ruptures the elastic fibers of the alveoli, aiding in the formation of the

TABLE II: SIMPLE SPONTANEOUS PNEUMOTHORAX

Number of Cases (1930–47)	97	
Sex		
Males	87	
Females	10	
Age		
Average	30.2	years
Youngest	17	years
Oldest	71	years
Race		
White	92	
Negro	5	
Location		
Left	44	
Right	47	
Bilateral	6	
Amount of Collapse		
Over 50%	57	
Under 50%	31	
Hydrothorax	46	
Hemothorax, Massive	3	
Thoracentesis	9	
Re-expansion		
Average	4	weeks
Shortest	6	days
Longest	12	weeks
Recurrences		
One	16	
Two or more	8	
Time Between Attacks		
Average	23.2	months
1 year or less	15	cases
5 years or more	5	cases

bulla. Active, robust men are most likely to overdistend their upper lobes. The glottis is closed in severe exertion, with the abdominal and expiratory muscles forcing the air into the upper lobes. The bullae may form in lungs free of disease or subpleural scar. The formation of the bulla is the first step in the pathogenesis. The next requirement is the establishment of a check-valve mechanism, which has been described by Hayashi. The entrance of air into the bulla is not impeded, but the egress of the air can thus be prevented. The bulla may continue to enlarge and thin out to such an extent that it cannot resist further increase in pressure, and will burst. Therefore, the development of simple spontaneous pneumothorax requires the association of all three factors (subpleural bulla, check-valve, and exertion). The lack of any one may either delay or prevent the occurrence of pneumothorax.

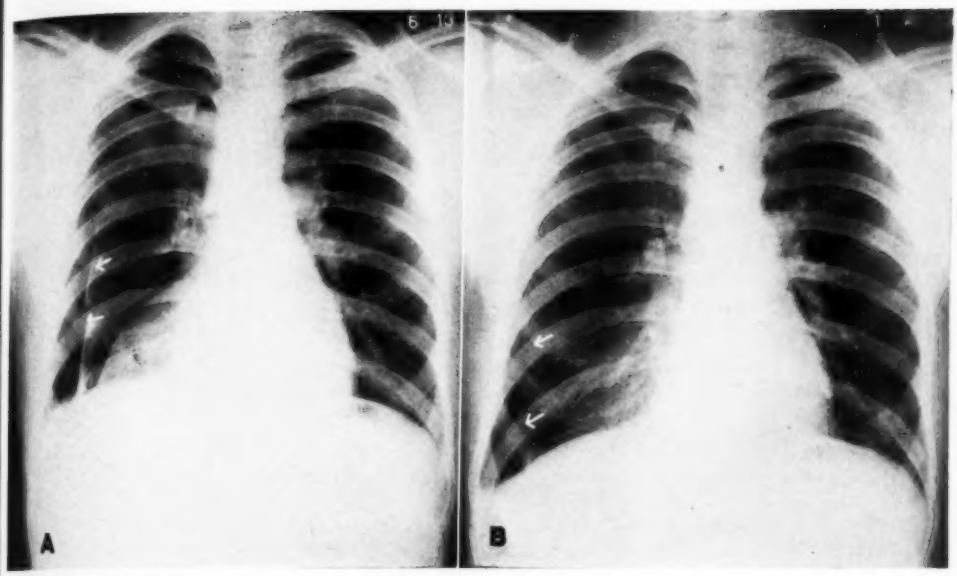
CLINICAL FEATURES

This report is based on 97 consecutive cases of simple spontaneous pneumothorax (exclusive of 8 cases in which the spontaneous pneumothorax was complicating a current intrathoracic disease process—6 cases of pulmonary tuberculosis, 1 of pleural carcinomatosis, and 1 of suppurative pleurisy).

Of the 97 patients, 49 have been observed for more than one year. Of the 49, 15 have been followed five to ten years, and 7 followed ten to eighteen years after the initial pneumothorax.

Investigators have noted the prevalence of simple spontaneous pneumothorax in young, active, robust men. Our statistics do not materially differ from previous observations. Eighty-seven of our cases occurred in men, and 10 in women. The average age was 30.2 years, with the youngest patient seventeen and the oldest seventy-one years. The association of exertion with the precipitation of the pneumothorax was notable for its infrequency. Most of our patients were not engaged in strenuous activity at the time of the attack. Usually, they were either in bed, sitting in a chair, or walking. Nine of the patients did give a history of exertion, which varied from jumping out of bed, running leisurely, severe coughing spell, to lifting heavy objects. A 51-year-old man experienced his attack following a flurry of manual labor in preparation for a hunting trip. The occupation of most of the patients was of a sedentary nature. One medical student, four interns and residents, and a bacteriologist are included in this group, all of whom were men. It is of interest that not a single case of spontaneous pneumothorax occurred among 14,000 student nurses who were trained at the Columbia-Presbyterian Medical Center between 1930 and 1947.

The infrequency of spontaneous pneumothorax among U. S. Air Force personnel was observed by Leach. Of the 126 cases reported by him, only 3 were associated with low-pressure chambers, and he con-



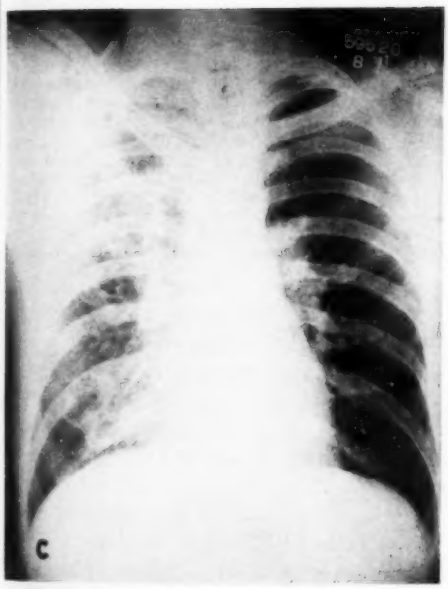


Fig. 1. Pulmonary tuberculosis developing fourteen years after spontaneous pneumothorax. A. Spontaneous pneumothorax with no evidence of tuberculosis, May 13, 1930. B. Second attack of spontaneous pneumothorax Jan. 21, 1931, with no evidence of tuberculosis. C. Tuberculosis of the right upper lobe with cavitation, Aug. 11, 1944.

G. M., a 26-year-old man, was admitted in May 1930 because of dyspnea and severe knife-like pain, aggravated by inspiration and motion, in the right side of the chest. He had night sweats twice a week for a year in 1925, and "pleurisy" on the right in 1929. He denied having easy fatigability, cough, hemoptysis, or tuberculosis in the family. Nine examinations failed to reveal acid-fast organisms in the sputum. Roentgen examination, May 13, 1930, disclosed pneumothorax on the right (A) with moderate collapse of the lung and a very small amount of fluid. No evidence of pulmonary tuberculosis was found on the film. Seventeen days later the lung was almost completely re-expanded.

In January 1931, the right pneumothorax recurred (B). Information obtained from another hospital disclosed that the patient experienced pain in the right chest in 1939. and a Board of Health roentgenogram revealed "a small cavity in the right lung." The patient felt well and continued working as a taxi driver until July 1944.

In July 1944, fourteen years after the first spontaneous pneumothorax, the patient complained of night sweats, fever, slight pain in the upper part of the right side of the chest, and anorexia. In six weeks he lost 18 lb. A roentgenogram of the chest, Aug. 11, 1944, disclosed evidence of tuberculosis with cavitation in the right upper lobe (C). The patient was transferred to a tuberculosis sanatorium, and tubercle bacilli were demonstrated in his sputum. He died on April 27, 1945.

sidered this association coincidental. No case of pneumothorax occurred during aerial flight.

The *U. S. Army Medical Bulletin* had a census of 873 cases of spontaneous pneumothorax hospitalized in continental United States in 1943. In about 15 per cent of the soldiers concerned, the disability was considered of sufficient gravity to warrant separation from the service.

The two major symptoms experienced by all our patients were chest pain and dysp-

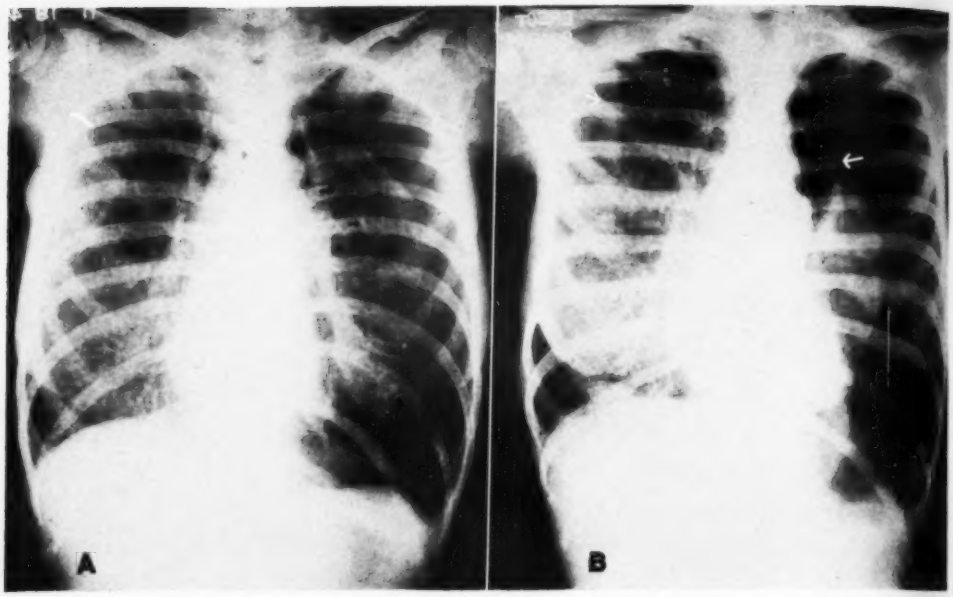


Fig. 2. Tension pneumothorax with pulmonary fibrosis, bullous emphysema, apical adhesions, and failure of re-expansion. A. Nov. 18, 1942. B. March 17, 1943.

M. C., a 46-year-old woman, had asthma for several years. During 1941 and 1942, she had several attacks of spontaneous pneumothorax, with incomplete re-expansion. She was admitted in November 1942 for study. Roentgenogram of the chest, Nov. 18, 1942 (A), showed left pneumothorax with collapse of the lung to less than half its normal size. The apex was attached to the chest wall by a long adhesion. The mediastinum was displaced slightly to the right, indicating increased pressure in the left hemithorax. Evidence of pulmonary fibrosis was present in both lungs, with large bullae in the left lung. Aspiration of air from the left side of the chest was not successful in expanding the left lung. After discharge, the dyspnea increased. A film of the chest on March 17, 1943 (B) showed still more collapse of the left lung and more displacement of the mediastinum to the right, and a partial pneumothorax on the right. Because of the increasing dyspnea, a thoracotomy tube was inserted into the left hemithorax to provide a constant outlet for the air. About ten minutes later the patient went into shock and died. Autopsy was not obtained. It was assumed that the pleural adhesion prevented the closure of a ruptured bulla.

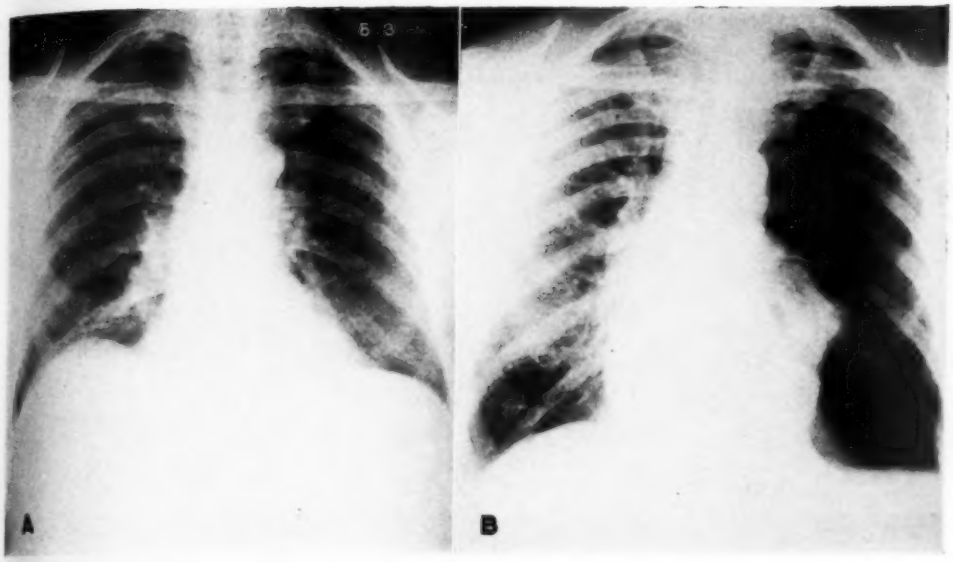
nea. The former was limited to the side involved, and often radiated to the shoulder, back, or abdomen. The pain came suddenly, and usually was severe enough to terrify the patient. The precordial pain simulated angina and augmented the apprehension. The pain usually subsided or disappeared within one to two days. The dyspnea also disappeared after a few days of bed rest, if no complications were present. Adhesions which prevented collapse of the bulla and permitted a patent bronchopleural system would result in persistent dyspnea. Nine of our cases required thoracentesis and removal of air from the thorax for relief from the dyspnea.

A low-grade fever was present occasionally at the onset. Cough was not a constant finding, and hemoptysis did not occur.

The right hemithorax was involved in 47 cases, with 44 cases occurring on the left, and 6 were bilateral. In 57 patients the lung collapsed to less than half its normal size and in 31 to more than half. Nine cases had no record of the degree of collapse.

Forty-six (47 per cent) cases had roentgenographic evidence of slight to moderate amounts of pleural effusion, which was gradually and spontaneously resorbed. The presence of fluid had been considered erroneously by many observers in the past as evidence in favor of tuberculosis. Our findings are more in agreement with those of Ornstein and Lercher, who reported an incidence of 38 per cent.

Massive hemothorax developed in three patients, requiring thoracentesis.



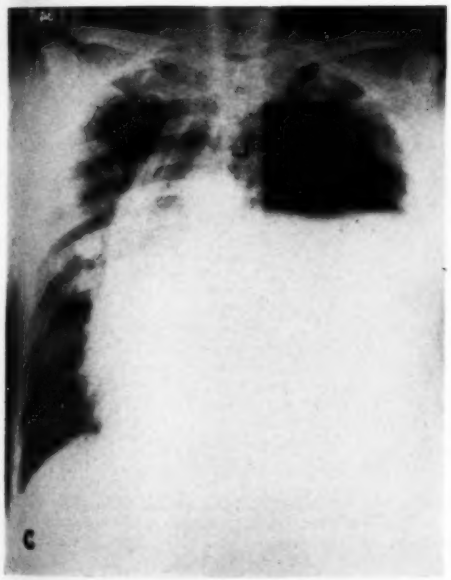


Fig. 3. Spontaneous pneumothorax with hemothorax. A. Roentgenogram of chest taken on May 31, 1932; the patient complained of anginal pains. B. Spontaneous pneumothorax, left, Sept. 25, 1934, with practically complete collapse of left lung and a small amount of fluid at left base. C. Spontaneous pneumothorax with large collection of fluid and contralateral mediastinal displacement, Nov. 7, 1934.

J. P., a 71-year-old man, was admitted because of a "squeezing" precordial pain of sudden onset which was thought by his family doctor to be of cardiac origin. For several years he had symptoms consistent with angina pectoris, pulmonary emphysema, and chronic bronchitis. A film of the chest Sept. 25, 1934 (B) disclosed a left pneumothorax with practically complete collapse of the left lung and a small amount of fluid at the left base. The fluid steadily increased, and on Nov. 7, 1934 (C) a large amount of fluid and mediastinal displacement were noted. The left side of the chest was aspirated several times for the relief of dyspnea, and air and grossly bloody fluid were removed. No tubercle bacilli or tumor cells were demonstrated in the fluid. Endoscopic examination of the left pleural cavity showed numerous hemorrhages in the parietal pleura. Six weeks after admission the patient died. Permission for autopsy was not obtained. The cause of the pleural hemorrhages was not determined.

The average time required for apparently complete re-expansion was four weeks. The shortest was six days, and the longest twelve weeks. The larger the pneumothorax, the longer was the time necessary for re-expansion.

Sixteen patients had one recurrence, and 8 had two or more. The average time between attacks was 23.2 months. Fifteen patients had recurrences in one year or less, and 5 in five or more years after the initial pneumothorax. As described by other investigators, recurrent pneumothorax is most likely to appear within one year.

Five of the patients disclosed evidence of pleural adhesions with fibrous bands. One patient had adhesions between the upper lobe and anterior chest wall, as well

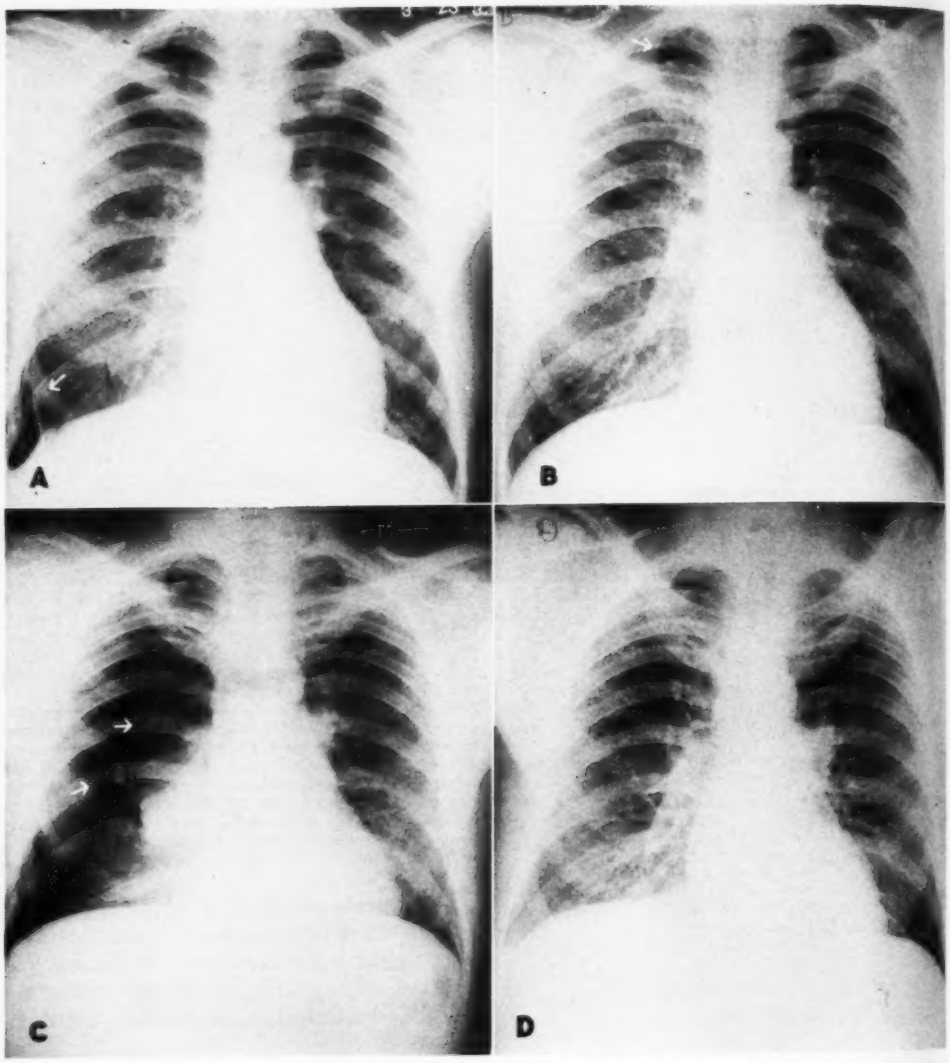


Fig. 4. Recurrent spontaneous pneumothorax, with demonstrable bullae. A. March 23, 1932, partial pneumothorax, right. B. May 21, 1940, bullae at right apex. C. Nov. 20, 1941, recurrent pneumothorax, right, with demonstrable bullae at apex of collapsed right upper lobe. D. Jan. 12, 1942. Roentgenogram of chest, patient asymptomatic.

J. R., a 36-year-old man, was admitted to the Presbyterian Hospital in 1932, 1934, and 1941 for spontaneous pneumothorax involving the right hemithorax. Re-expansion occurred spontaneously on each occasion. The patient was seen in 1947 (fifteen years after the initial pneumothorax) and appeared in excellent health.

as pleuropericardial adhesions. He was observed for only six months, during which time no complications were noted. The second patient disclosed adhesive bands between the upper lobe of the left lung and chest wall, and experienced three attacks of pneumothorax in a ten-year period. Bullae were observed roentgenographically. No clinical or laboratory evidence of pulmonary tuberculosis has been manifest. The third patient died three years after the initial pneumothorax, and obsolete, bilateral pulmonary tuberculosis was found at autopsy. In the

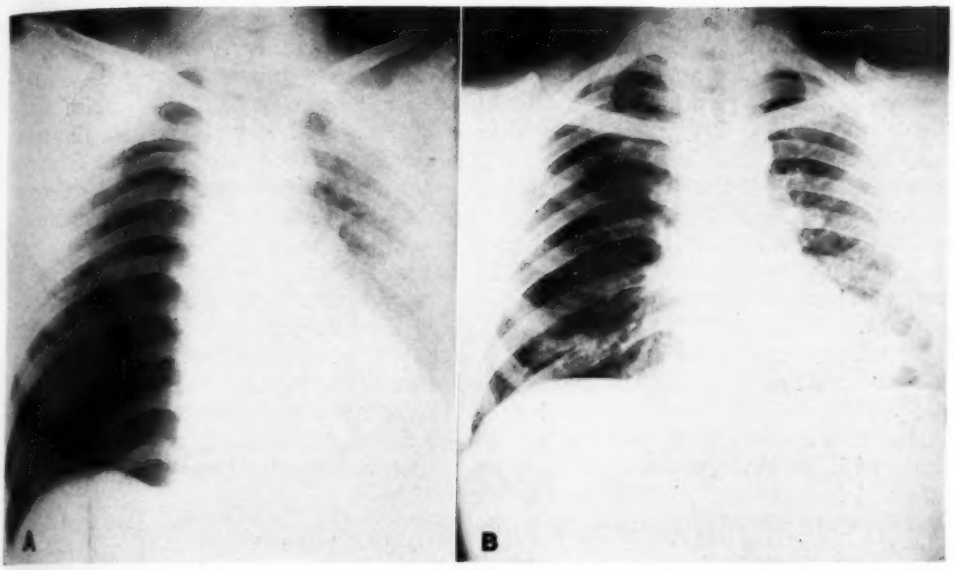


Fig. 5. Simple spontaneous pneumothorax treated as a case of pulmonary tuberculosis. A. Spontaneous pneumothorax, right, tension type, with left lateral mediastinal displacement, March 28, 1940. B. Roentgenogram of chest on April 18, 1940, following five aspirations for relief of dyspnea, disclosing over 75 per cent re-expansion of right lung. No evidence of tuberculosis.

J. D., a 30-year-old white man, a clerk in the employ of a large life insurance company, experienced a spontaneous pneumothorax on the left in 1933. This attack was assumed to be the result of tuberculosis, and the patient was treated in a sanatorium from June 1933 to March 1935. He remained symptom-free, and repeated sputum examinations were negative for tubercle bacilli. During his stay in the sanatorium he experienced the second and third attacks of spontaneous pneumothorax, again involving the left side, in December 1933 and July 1934. At this latter date artificial pneumothorax was administered, and maintained from July 1934 to January 1935, when re-expansion was permitted, which was followed by another (fourth) spontaneous pneumothorax. Bronchoscopy was performed in June 1936, due to incomplete re-expansion of the lung, and was reported as negative. In November 1939 the fifth spontaneous pneumothorax developed following a cough, involving the right side for the first time. The right lung was entirely collapsed, and very slowly and incompletely re-expanded.

On March 28, 1940, the patient was hospitalized at the Presbyterian Hospital for his sixth spontaneous pneumothorax. His major complaint was dyspnea. A roentgenogram of his chest (A) on the day of admission disclosed a tension pneumothorax on the right, with left lateral displacement of trachea and heart, and mediastinal herniation of the right lung into the inferior portion of the left hemithorax. Suggestive roentgenographic evidence of bullae was noted on later films.

The patient required five aspirations during this hospital stay for relief of his dyspnea produced by the tension pneumothorax. Due to the possibility of pneumonitis at the left base, sulfonamides were administered. Within twenty-one days the right lung showed over 75 per cent re-expansion. No evidence of pulmonary tuberculosis was demonstrated roentgenographically or by laboratory procedures.

fourth case autopsy disclosed no obvious cause for the adhesive band. The fifth patient had night sweats in 1925, "pleurisy" in 1929, spontaneous pneumothorax in 1930, and active pulmonary tuberculosis in 1944. It is not entirely clear whether this last patient had pulmonary tuberculosis before his initial attack of spontaneous pneumothorax.

Ornstein and Lercher reported in their series of 58 cases that in 3 patients with pleural adhesions pulmonary tuberculosis developed within two years. They suggested that pleural adhesions must be taken as a warning that tuberculosis may ensue. Our experience approximates that of Kjaergaard, who observed 6 cases with pleural adhesions in none of whom tuberculosis developed.

PROGNOSIS

The immediate prognosis in simple spontaneous pneumothorax (excluding complications) is excellent. Kjaergaard had no deaths in 51 cases. Perry had one death in 85 cases, in a patient with a concurrent septicemia following a whitlow.

The forms of the disease which involve

a danger to life are tension pneumothorax, hemopneumothorax, and bilateral pneumothorax. Two deaths occurred in our series. Tension pneumothorax resulted in the death of one of our patients, with no relief by surgical intervention. A massive hemothorax caused the death of the other patient.

Up to 1939, 21 cases of hemothorax complicating spontaneous pneumothorax were reported, with a mortality rate of 37 per cent. Recently (1946) Bernstein, *et al.* reviewed the literature on spontaneous hemothorax, and was able to collect 44. Forty of these cases were of the hemopneumothorax variety, and 4 were unassociated with free air. Housden and Piggot's case had fibrous bands, which were found at autopsy to extend from the apex of the lung to the chest wall. They quote Pitt's case which, at autopsy, showed a torn emphysematous bulla attached to the fibrous band. No obviously patent vessel was located. They also quote Krause and Heise, who reported a case in which a man undergoing artificial pneumothorax died of a hemothorax due to the tearing of the pleural adhesion. It has been suggested that the vessels which grow into the adhesions are the source of the bleeding when the latter are torn.

Bilateral spontaneous pneumothorax occurred in 6 of our patients. Twenty cases of bilateral spontaneous pneumothorax were reported up to 1938, with 10 deaths (50 per cent mortality). The cases reported more recently appear to have a better prognosis.

Mediastinal emphysema did not occur in any of our cases.

DISCUSSION

The roentgenographic demonstration of subpleural bullae is frequently impossible. The presence or absence of tuberculosis is the diagnostic problem, and one must attempt to determine whether or not the existing pneumothorax is a complication of pulmonary tuberculosis. Spontaneous pneumothorax constitutes about 3 per cent of the immediate causes of death among tuberculous patients at the Montefiore Hospital (Fishberg).

Pulmonary tuberculosis as the etiologic agent must be ruled out by clinical investigation. Fishberg states that the development of spontaneous pneumothorax in very acute pulmonary tuberculosis results in a 90 per cent mortality rate within one month. The negative tuberculin test, normal sedimentation rate, absence of fever and pyothorax, and the prompt recovery without sequelae are the most reliable criteria in the differential diagnosis. If tuberculosis is not present at the time the pneumothorax occurred, it is no more likely to develop than in the average individual in the community (Perry).

SUMMARY

This report is based upon 105 consecutive cases of spontaneous pneumothorax observed at the Columbia-Presbyterian Medical Center, New York, from 1930 to 1947, inclusive. Ninety-seven occurred in apparently healthy individuals. The average age was 30.2 years, and the great majority were males. Roentgenographic evidence of pleural fluid was present in 47 per cent of the cases. This study indicates that pulmonary tuberculosis, if not present at the time of the attack, rarely developed subsequent to the spontaneous pneumothorax.

Presbyterian Hospital
622 W. 168th St.
New York 32, N. Y.

BIBLIOGRAPHY

BABINGTON, H. S.: Idiopathic Spontaneous Pneumothorax. West. J. Surg. **52:** 73–76, 1944.

BERNSTEIN, A., KLOSK, E., AND PARSONNET, A. E.: Spontaneous Hemothorax. Dis. of Chest **12:** 394–401, 1946.

BLACKFORD, S. D.: Spontaneous Pneumothorax in College Students. J. A. M. A. **113:** 737–739, 1939.

BROCK, R. C.: Recurrent and Chronic Spontaneous Pneumothorax. Thorax **3:** 88–111, 1948.

Bull. U. S. Army M. Dept. (no. 82) p. 29, 1944.

CAFFEY, JOHN: Personal communication.

FISCHER, B.: Der gutartige Spontanpneumothorax durch Ruptur von Spitzennarbenblasen, ein typisches Krankheitsbild; mit Beiträgen zur Lehre vom Emphysem. Ztschr. f. klin. Med. **95:** 1–50, 1922. Cited by Kjaergaard, 1932.

FISHBERG, M.: Pulmonary Tuberculosis, Philadelphia, Lea & Febiger, 1932. Vol. II, p. 105.

FOLKE, L.: Persistent Spontaneous Pneumothorax in Infants. Acta pædiat. **17:** 426–438, 1935.

GOUGH, J.: Fatal Pneumothorax Due to Rupture of a Solitary Bulla of the Lung. Lancet **2:** 314–315, 1937.

HAMMAN, L.: Spontaneous Pneumothorax. Am. J. M. Sc. **151:** 229–249, 1916.

HAYASHI, J.: Über totlichen Pneumothorax durch Infarkt und Emphysem. Frankfurt. Ztschr. f. Path. **16:** 1–36, 1914. Cited by Kjaergaard, 1932.

HEATH, E. M.: Spontaneous Pneumothorax in Healthy Young Adults. Am. J. M. Sc. **211:** 138–143, 1946.

HELWIG, F. C., AND SCHMIDT, E. C. H.: Fatal Spontaneous Hemopneumothorax. Ann. Int. Med. **26:** 608–617, 1947.

HOUSDEN, E. G., AND PIGGOT, A.: Spontaneous Haemopneumothorax with Unusual Postmortem Findings. Brit. M. J. **2:** 941–943, 1931.

HYDE, B., AND HYDE, L.: Benign Idiopathic Spontaneous Pneumothorax. Am. J. M. Sc. **215:** 427–430, 1948.

ITARD, J.: Sur le pneumothorax ou les congestions gazeuses qui se forment dans la poitrine. Thesis, Paris, 1803. Cited by Kirshner, 1938.

KIRSHNER, J. J.: Spontaneous Pneumothorax. Am. J. M. Sc. **196:** 704–708, 1938.

KJAERGAARD, H.: Spontaneous Pneumothorax in the Apparently Healthy. Acta med. Scandinav. supp. 43, pp. 1–159; 1–93, 1932.

KJAERGAARD, H.: Pneumothorax Simplex. Two Cases with Autopsy Findings. Acta med. Scandinav. **80:** 93–104, 1933.

KJAERGAARD, H.: Cystic Lungs. Acta med. Scandinav. **86:** 407–434, 1935.

LEACH, J. E.: Pneumothorax in Young Adult Males. Arch. Int. Med. **76:** 264–268, 1945.

MCDOWELL: On an Unusual Form of Pneumothorax. Dublin Hosp. Gaz. **3:** 227–229, 1856. Cited by Kjaergaard, 1932.

MILLER, W. S.: The Lung. Springfield, Ill., Charles C Thomas, 2nd ed., 1947, Chap. IX.

MYERSON, R. M.: Spontaneous Pneumothorax. New England J. Med. **238:** 461–463, 1948.

NIEHAUS, R. F.: Simple Spontaneous Pneumothorax in Apparently Healthy Individuals. Am. J. Roentgenol. **57:** 12–27, 1947.

ORNSTEIN, G. G., AND LERCHER, L.: Spontaneous Pneumothorax in Apparently Healthy Individuals. Quart. Bull., Sea View Hospital **7:** 149–187, 1942.

PEASE, P. P., STEUER, L. G., AND CHAPMAN, A.: Spontaneous Pneumothorax in Soldiers. Bull. U. S. Army M. Dept. (no. 82) pp. 102–107, 1944.

PERRY, K. M. A.: On Spontaneous Pneumothorax. Quart. J. Med. **8:** 1–21, 1939.

PITT, G. N.: Case of Rapidly Fatal Haemopneumothorax Apparently Due to the Rupture of an Emphysematous Bulla. Tr. Clin. Soc. London **33:** 95–115, 1899–1900. Cited by Perry, 1939.

RUBIN, E. H.: Diseases of the Chest, with Emphasis on X-Ray Diagnosis. Philadelphia, W. B. Saunders Co., 1947.

SCHMINCKE, A.: Zur Genese des doppelseitigen Spontanpneumothorax. (Zugleich ein Beitrag zu den Missbildungen des Lungengewebes). Beitr. z. path. Anat. u. z. allg. Path. **80:** 692–696, 1928.

SCHNEIDER, L., AND REISSMAN, I. I.: Idiopathic Spontaneous Pneumothorax. Radiology **44:** 485–488, 1945.

SOKOLOFF, M. J., AND FARRELL, J. T., JR.: Spontaneous Pneumothorax in Anthracosilicosis. J. A. M. A. **112:** 1564–1566, 1939.

STEIN, G. H., MCCONKIE, E. B., AND KUEHN, A. J.: Spontaneous Pneumothorax. War Med. **4:** 324–330, 1943.

TASCHMAN, M.: Spontaneous Pneumothorax. J. Mt. Sinai Hosp. **10:** 684–697, 1944.

TYSON, M. D., AND CRANDALL, W. B.: Surgical Treatment of Recurrent Idiopathic Spontaneous Pneumothorax. J. Thoracic Surg. **10:** 566–571, 1941.

VAN ORDSTRAND, H. S.: Idiopathic Spontaneous Pneumothorax. Cleveland Clinic Quart. **7:** 178–183, 1940.

WEST, S.: Case of Complete Recovery from Pneumothorax without Effusion of Fluid. Tr. Clin. Soc. London **17:** 56–59, 1884. Cited by Perry, 1939.

WILLCOX, A., AND FOSTER-CARTER, A. F.: Spontaneous Pneumothorax Associated with Bullous Emphysema. Lancet **2:** 315–317, 1937.

WOOD, H. G.: Bilateral Spontaneous Pneumothorax. Minnesota Med. **14:** 550–552, 1931.

SUMARIO

Neumotórax Espontáneo. Estudio de 105 Casos

El repaso de la literatura muestra que la patogenia del neumotórax espontáneo es atribuída por varios investigadores a tres importantes factores etiológicos: tuberculosis pulmonar, vesículas congénitas y enfisema.

La actual comunicación tiene por base 105 casos consecutivos de neumotórax espontáneo, observados en el Centro Médico Columbia-Presbyterian de Nueva York, de 1930 a 1947, inclusivo. Noventa y siete ocurrieron en individuos aparentemente sanos. La edad promedió 30.2 anos, y la inmensa mayoría de los enfermos fueron varones. En 47 por ciento había signos roentgenográficos de derrame pleural. Este estudio indica que, sino existe ya para la fecha del ataque, rara vez se presenta tuberculosis pulmonar después del neumotórax espontáneo.

Bronchial Dynamism[1]

DR. S. Di RIENZO
Córdoba, Argentina

THE BRONCHI ARE not simply "air passages" or air conduits. They possess anatomic and physiologic characteristics destined to fulfill a much more important function than that of mere air-distributing channels. But these vital characteristics must not be sought in the bronchial cast, for not even traces of them are to be found in the cadaver, and many of them are not even detected by direct bronchoscopic examination. They are probably best demonstrated by roentgenography after the introduction of a contrast medium, but a single picture taken after the opacification of the bronchial tree is not sufficient to afford a definite knowledge of its functional mechanism. It is necessary to introduce the opaque medium under fluoroscopic guidance, to instruct the patient as to inspiration, expiration, and coughing, and to obtain multiple views—serial and spot films—during the various respiratory processes. It is only thus that the complicated dynamic functions fulfilled by the bronchi may be learned; and upon this physiopathologic basis we may build up a theory of the pathogenesis of the respiratory syndromes.

This method of radiologic investigation allows us to determine the anatomicophysiological characteristics of the normal bronchus. These characteristics are indicated chiefly by: (1) the rhythm and manner of filling; (2) the variations of caliber and static and dynamic sphincteric alterations; (3) the results of pharmacodynamic tests.

Rhythm and Manner of Filling: Fluoroscopically, one observes, while injecting iodized oil into the air tubes, that without question the opaque medium flows into the trachea and into the major bronchi (right and left stem) by the action of gravity. Recumbency has a decisive effect on the route followed by the oil in these major tubes, whereas the influence of the respiratory movements is nil. These facts hold true so long as the quantity of opaque medium is not sufficient to obstruct the tracheobronchial space, for in the latter event the flow is influenced also by the thrust of the column of air that seeks to force its way into or out of the lung.

When the opaque medium reaches the first subdivisions of the main bronchi, that is, the secondary or lobar bronchi, it is noticed that the flow is not progressively continuous as when under the influence of a constant force of invariable magnitude, but that it is pulsating and rhythmic with the respiration. Fluoroscopically this difference between the progressive, continuous flow in the large bronchus and the progressive rhythmical flow in the lobar branches is clearly manifest.

When the opaque medium reaches the branches of still smaller caliber—the bronchioles—its progress is slow and is limited to the inspiratory phase. The flow now shows a forward and backward movement, the oil advancing during inspiration and partially receding during expiration. Under normal conditions the inspiratory advance is noticeably greater than the expiratory recession, but in pathologic cases these movements may be absent or the two may be equal, thus preventing the introduction of the oil into the bronchioles and into the alveoli.

This inspiratory progress of the opaque medium is due solely to thoracic-alveolar aspiration, which is of unsuspected magni-

[1] Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

Acknowledgement is here made of the courtesy of Charles C Thomas, Springfield, Ill., for permission to reproduce many of the illustrations. These will appear in Dr. Di Rienzo's book "Radiologic Exploration of the Bronchus," Springfield, Ill., Charles C Thomas, 1949.

tude. If we attempt to pass iodized oil (50 per cent) through a glass tube of the same caliber as the bronchiole we will see that, even favored by gravity, it progresses either very slowly or not at all, in contrast to what occurs in the lung, where, at each inspiration, the opaque medium advances rapidly as if there were no obstacle. In fact, the result in the tube is what might be expected if the viscosity of the oil were five times as great as of that in the bronchus. To repeat, the advance of the oil in the bronchioles is due to the thoracic-alveolar aspiration, and for this reason every factor that alters that process retards or prevents the advance of the opaque medium. This is what happens in the presence of pleuropulmonary consolidation and sclerosis, in pleurisy, in peribronchial pneumonitis or bronchiectasis, and even when some painful thoracic or abdominal condition prevents the expansion of the thorax or the normal movement of the diaphragm.

The filling of the bronchial canal must be uniform: the opaque column must not be interrupted or contain any air or secretion which produces bubbles. If, however, bubbles are few in number and of brief duration, while other conditions are normal, they are not to be regarded as pathological; they are due to the fusion of multiple small bubbles released from the finer branches, which, having coalesced, are trying to find a way to the surface of the oil.

The fine secondary branching must appear simultaneously throughout the pulmonary zone to which the opaque medium has penetrated, or in the order in which the medium reaches the main bronchus. In general, this finer branching is evident earlier in the lower parts of the pulmonary zones, because the thoracic-alveolar aspiration is more energetic there, due to the great movement caused by diaphragmatic displacement. These zones, furthermore, are favored by the law of gravity.

The temperature and viscosity of the opaque medium employed play here an important role, for the caliber of the secondary branches is very narrow. The viscosity index of the liquid that must pass through them therefore conditions the velocity of its advance. Cold oils containing large opaque molecules have a high viscosity, so that they enter the narrow canals with great difficulty. Warm oils, on the other hand, containing opaque molecules of small diameter, are less viscous and pass quickly into the narrow bronchi.

Attempts to indicate a normal time during which the opaque medium should reach the final branches, that is, going all the way from the main bronchus to the alveoli, are of no practical value. Diverse factors may intervene to produce considerable variations in this respect which are of no pathological significance. The continual succession of bronchial images from the time the opaque medium is injected into the main bronchus until it reaches the alveolus is the best sign of normalcy. The time of succession of these images has not the importance that has been attributed to it, for it is conditioned by extrabronchial factors which are mostly technical.

The main point to be borne in mind is that the bronchographic images are normally changeable, transitory, and nonpermanent. When a succession of images does not occur in any one branch, it is because some pathologic factor has intervened and the dynamism of the branch has decreased or disappeared. Further on, we shall deal with the difference existing between the bronchographic aspect of the branch deprived of normal dynamism and that possessing it.

The normal bronchographic picture of the "foliage," as we may call the alveoli, is one of fine dots, which may be seen with the naked eye, or better with a lens. After a certain time, always very brief, this fine granulation becomes confluent and thickened. The "foliage" should appear simultaneously in every part which is reached by the opaque medium at the same time and must not be irregular in its characteristics.

The canalicular image differs from the alveolar image—that is, the branching

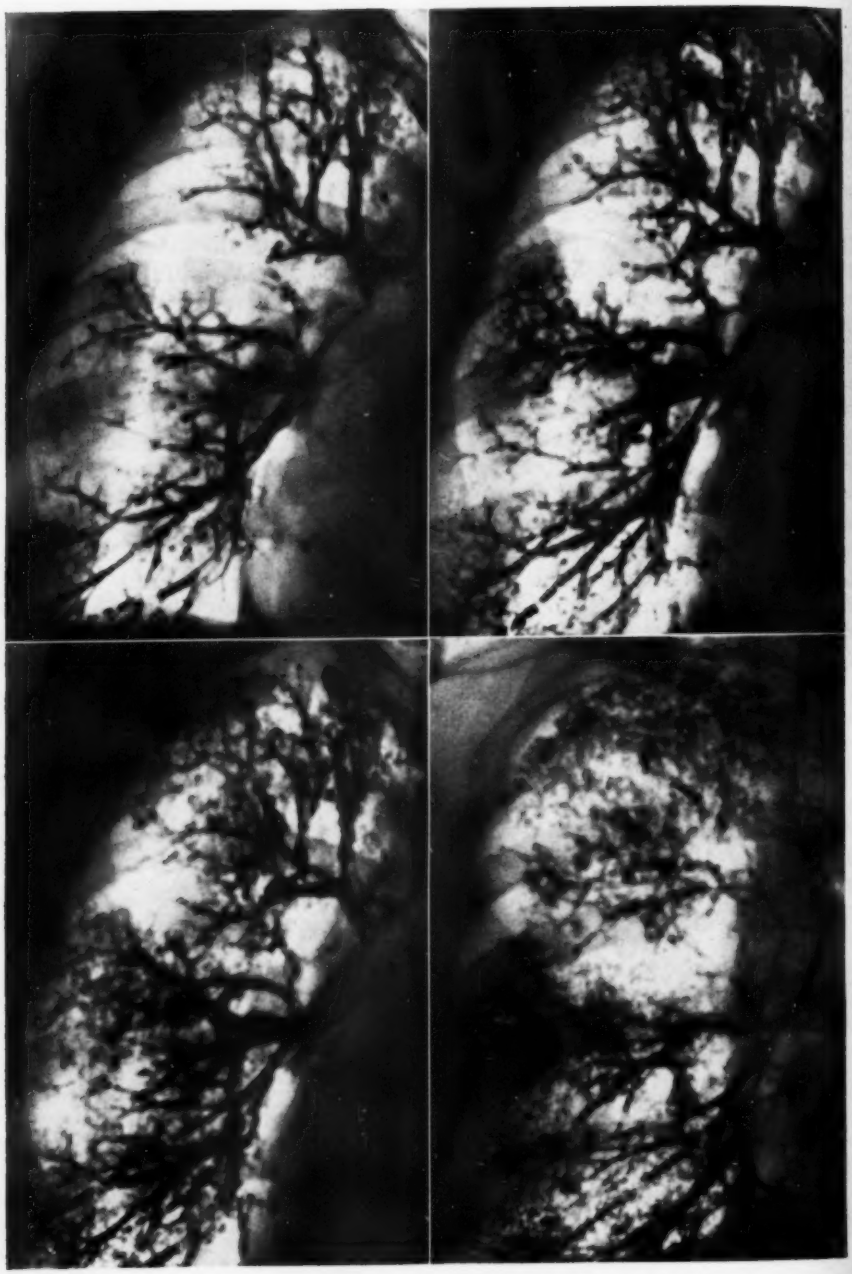


Fig. 1. Serial record of normal filling of the bronchial tree.

from the foliage—in that the former determines transitory images, while the latter determines permanent images. Once the image of the "foliage" is demonstrable, it does not disappear until after a few weeks or months, being more permanent in normal cases than in the presence of disease.

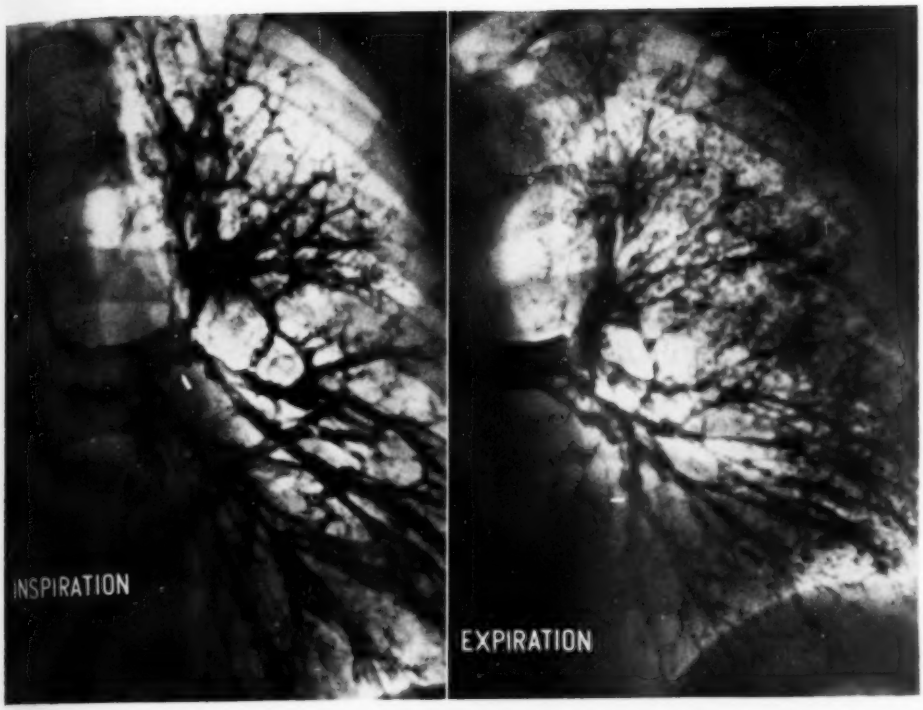


Fig. 2. General view during inspiration and expiration in a patient fifty years old with chronic bronchitis.

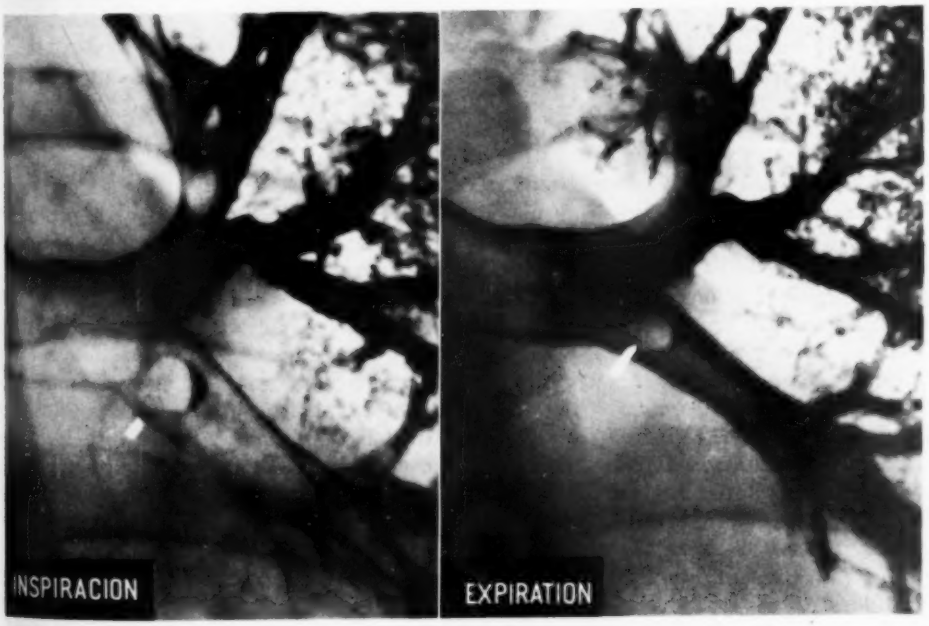


Fig. 3. Main bronchi during inspiration and expiration. Notice the reduction of caliber of the dorsal bronchus.

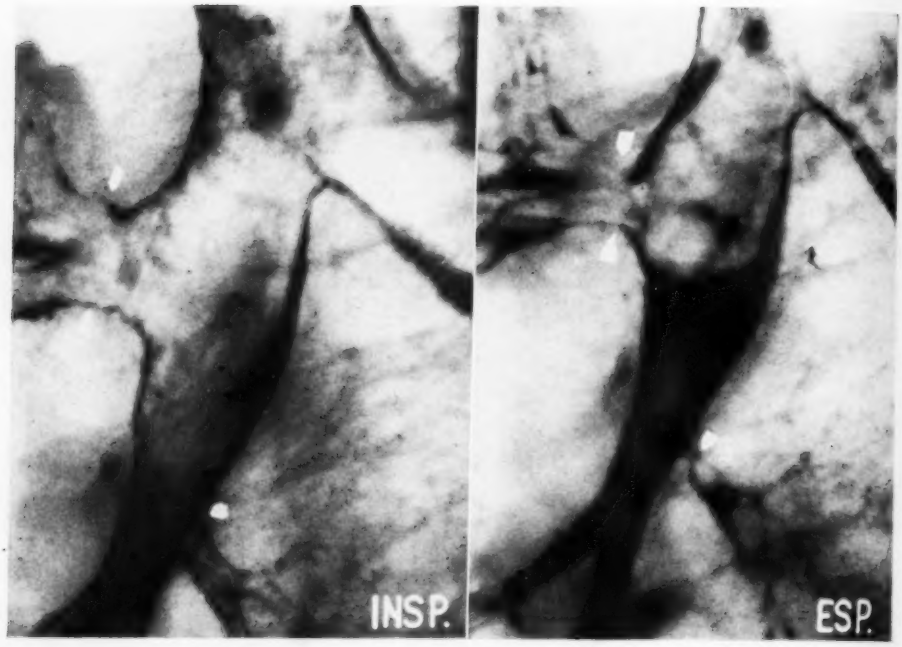


Fig. 4. Expansion of the bronchus during inspiration and contraction during expiration. Notice the expiratory stenosis of the main superior and first dorsal bronchus.

Variations of Caliber and Sphincteric Movements: The bronchi undergo variations in caliber, a knowledge of which is necessary to explain the respiratory syndromes. These variations are normal and pathologic and are associated with breathing, coughing, and crying. Modifications of the bronchial caliber may occur throughout the bronchus, that is to say they may be general, or they may be segmental or annular, the latter fulfilling the physiologic role of a sphincter, regulating the entrance and exit of air or secretions into the bronchial branches.

The changes of bronchial caliber are conditioned by the existence of smooth muscle fibers, elastic fibers, and nervous plexuses, which are located in the mucous membrane and in the wall of the bronchus. The smooth muscle fibers constitute a continuous layer beneath the outer wall of the bronchus as illustrated in Miller's schematic drawing, reproduced in Figure 5. At the root of each branch these fibers take the form of a slip-knot, swaddling the origin of the branch, and causing its strangulation when they are shortened in contraction.

The nervous system is constituted by plexuses which are disposed along the outside of the cartilage (extrachondral plexus), on the inner side of the cartilage (subchondral plexus), and in the epithelium (subepithelial plexus). The extrachondral plexus is attached to that of the vascular branch which lies next to the bronchus and its fibers proceed from the sympathetic ganglia, while those of the subchondral and the subepithelial plexuses are derived from the lower branches of the pneumogastric.

Normally the bronchial caliber changes during respiratory movement, increasing during inspiration and decreasing during expiration. These modifications occur in the large as well as in the finer bronchi. They are well seen during fluoroscopic examination and can be recorded in serial pictures, as shown in Figures 2 and 3. This inspiratory expansion and expiratory contraction are uniform all along the

bronchial wall, but at the end of expiration it is noticed that the reduction of the caliber is accentuated at the root of the branches, especially those of the second or third order. This contraction is very manifest in patients with chronic bronchitis, in those suffering from asthma or allergy, and in persons who have inhaled toxic gases. These bronchographic observations prove the existence of particular anatomic characteristics at the root of the bronchial branches and of a special innervation in this region. This disposition of the muscle fibers at the origin of the branches, as shown by Miller (Fig. 5), and the observation of the nerve ends in these regions, indicate the existence of a sphincter, chiefly in the main bronchi.

As we have already pointed out, these sphincters are clearly seen in the main lobar trunks, and for that reason we have come to call them *truncular sphincters*. Miller's anatomic studies show that similar arrangements exist in the alveolar ducts, and clinical studies present pictures determined by the closing of these end sphincters. That is why, although they are not susceptible of radiologic demonstration owing to their small size, we have come to give them also a name, designating them as *alveolar sphincters*, to indicate their extreme position, as compared to that of the truncular sphincters. Between these two functional sphincters there exist all those situated between branches of the third and fourth order.

The tone of these sphincters may be increased by (*a*) local causes, which play the role of "irritative thorns," like foreign bodies, in a cumulative way, and (*b*) by general causes, as happens in asthmatic patients or generally in the allergies. It is easily seen that the contraction at the root of the bronchus constitutes a retrograde obstacle in the ventilation of affected segments, with consequent retention of the secretions, followed by a functional and anatomic complex which masks the simple initial cause. On considering some asthmatic and bronchiectatic bronchograms, we shall again take up this important role of the sphincters that regulate the ventilation of the lung.

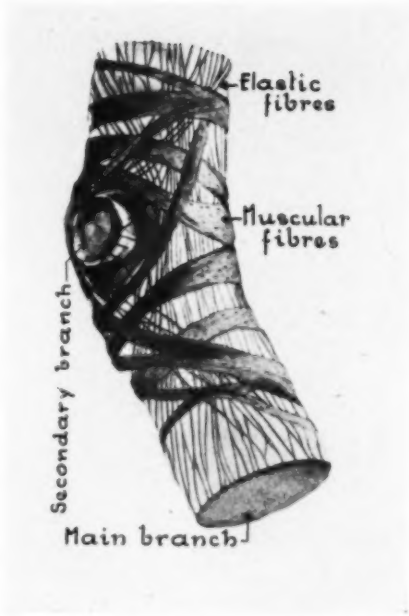


Fig. 5. Disposition of the smooth and elastic fibers at the root of the fine branches. (From The Lung, by W. S. Miller, Charles C Thomas, Springfield, Ill., 1947.)

Physiopathology of Cough: Radiographic records have allowed us to become acquainted with unsuspected aspects of the physiology of cough, leading us to regard it not as a simple act of expulsive hypertension, but as a dynamic act of the mucous membrane which expels the air or the secretions by means of a high-speed peristaltic wave originating in the small bronchi and ending in the vocal cords, accompanied by a harmonic movement of the functional sphincters. It may be compared to the act of vomiting. It is not the contraction of the abdomen which expels the gastric contents. It is an antiperistaltic wave of the stomach beginning next to the pylorus, by its closing, and ending next to the cardia by the opening of the sphincter. The abdominal hypertension favors vomiting, but does not cause it. In the bronchus, it is not the hypertension that expels the air and secretions during coughing; the air and secretions are ex-

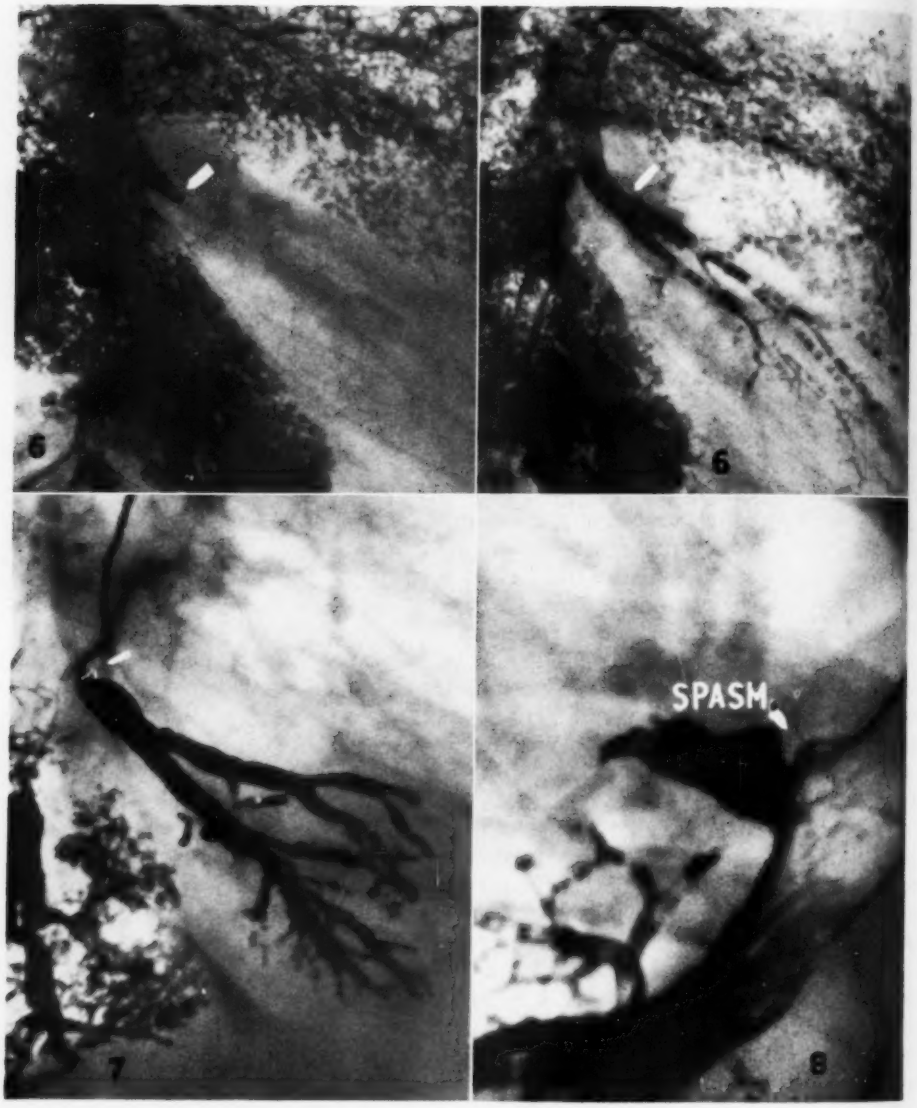


Fig. 6. Truncular spasm at the root of the middle lobe bronchus which has disappeared after local anesthesia of the affected bronchus.
Fig. 7. Spasm in the middle lobe bronchus.
Fig. 8. Spasm in the upper lobe bronchus.

pelled by the bronchus, as if they were foreign bodies, by a peristaltic expulsive wave.

Our conclusions are based on the fact that during the cough the bronchus does not bend or shrink, as would happen if the expulsion of air and secretions were due to a compression of the lung, comparable to compression of a sponge in the hand. On the contrary, at the beginning of the cough the bronchus stretches and rectifies itself as if it were in erection.

We have also observed that cough may take place separately in each lung, or even in each lobule, which means that there exist nerve centers independent of those

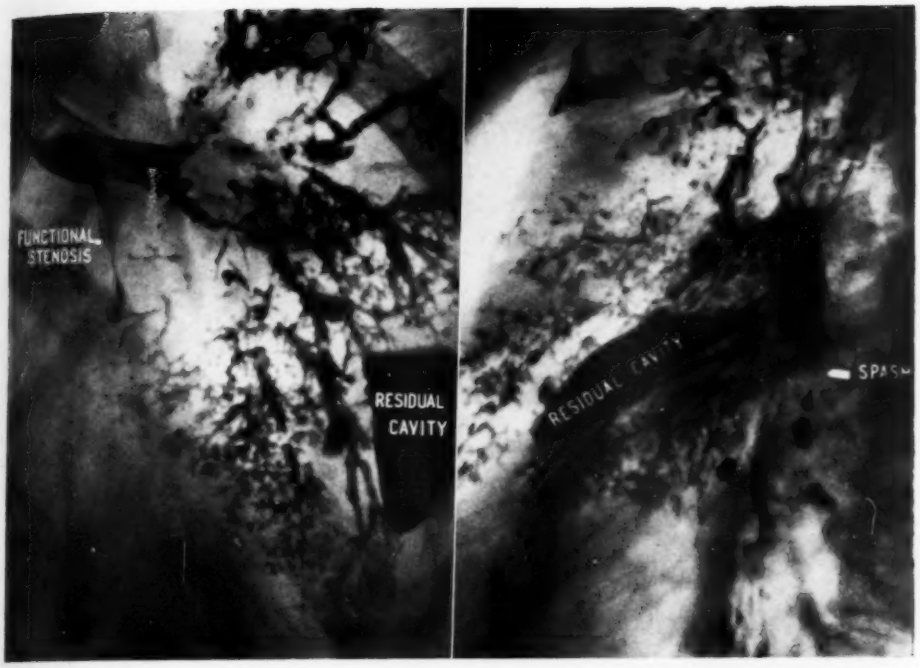


Fig. 9. Spasm in the main lower bronchus in a patient forty years old, after an operation for hydatid cyst.

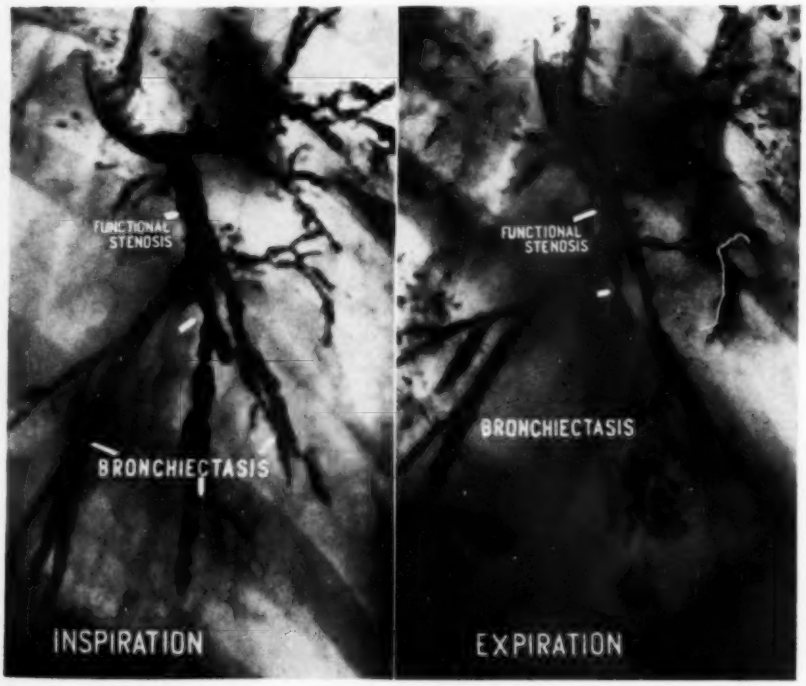


Fig. 10. Bronchograms of the same patient after anesthesia of the lower bronchus. Note the changes during inspiration and expiration.

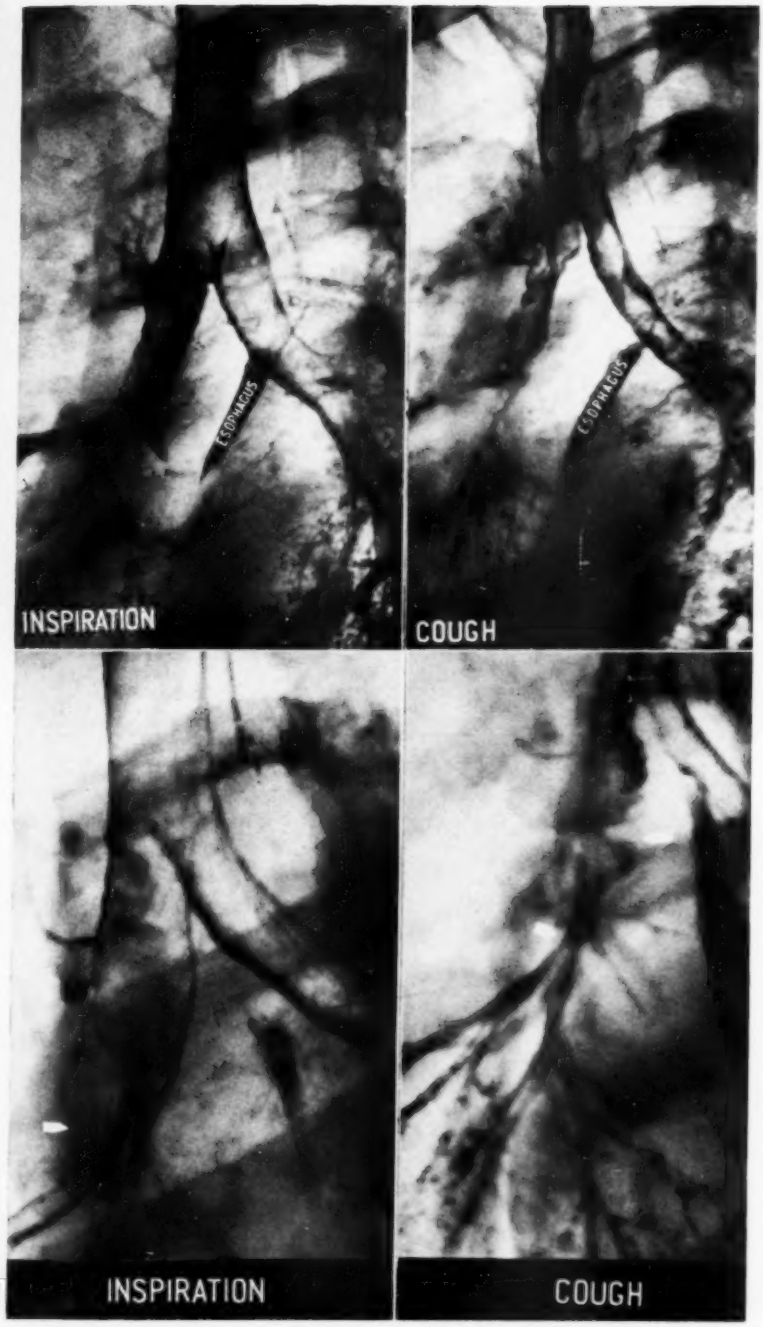
Fig. 11. Spasm of the fine bronchi as they appear in an asthmatic patient during a crisis. Notice strangulation of the right main bronchus.

that govern diaphragmatic contraction and the muscles of the thorax. Furthermore, bronchial branches which have been destroyed by infection or have been modified by bronchiectasis do not expel their contents during cough, for they have lost their dynamic properties.

Our observations on the various bronchopulmonary processes at different ages have convinced us that the physiologic act of coughing involves an important dynamic function and that it must not be interpreted solely as a reflex act determined by irritation of the bronchial tree, but as a mechanism of intelligent defense of the bronchopulmonary system.

In Figures 12 and 13 we show the aspect of the trachea and right bronchi during inspiration; a portion of opacified esophagus is also seen. In the same figure we reproduce what was observed in the same patient while coughing, keeping the technical conditions similar, to be able to make a true comparison. The comparative observations allow us to appreciate the substantial changes occurring in the bronchi and trachea during the act of coughing. We see that the right main trunk, as well as the inferior lobe bronchus, has wrinkled and retracted, producing undulations. The secondary branches have become filiform, having diminished up to a third of their normal caliber. But the strange thing is that all of this dynamic act, so manifest, has taken place solely in the bronchial tube; the esophageal tube has taken no part in it. It is clear that there has been absolutely no movement of the barium-filled esophagus, proving that the expulsive act of coughing has taken place in the bronchial wall and not within the whole thorax.

In Figure 14 we reproduce what occurs in the bronchial tree during inspiration, expiration, and coughing, in the same patient. In these three bronchograms we can see the great variations of the caliber of the bronchi and the accentuation of the



Figs. 12 and 13. Bronchograms taken during inspiration and coughing.

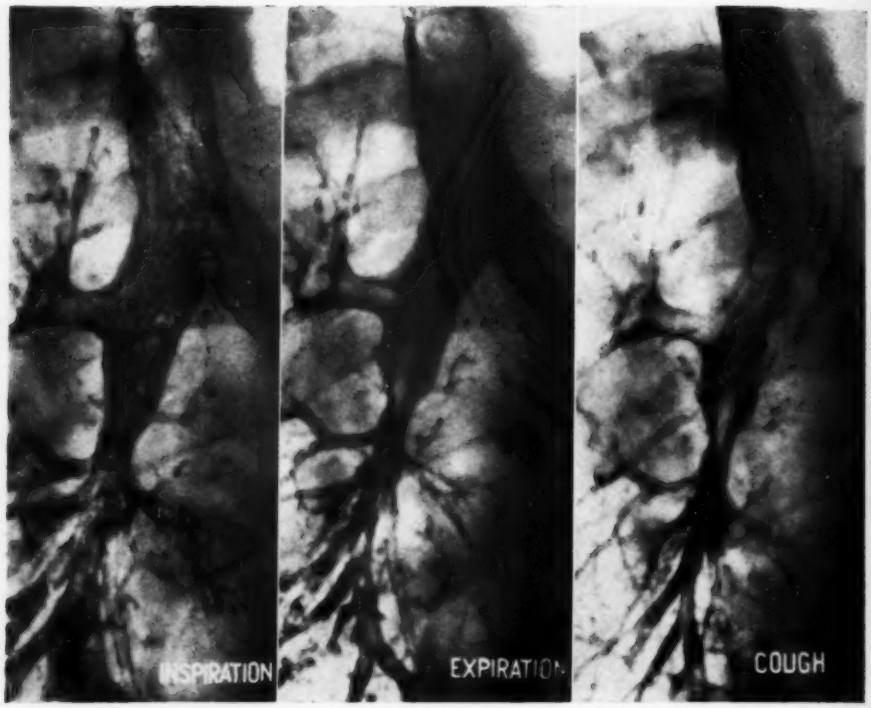


Fig. 14. Comparative study of the bronchial tree during inspiration, expiration, and cough.

sphincteric tonus which causes the closing of the right upper bronchus and that of the middle lobe. It is important to recall that it is in these very lobes that bronchiectasis occurs.

In Figure 15 is shown what takes place during inspiration and expiration, at the beginning of a cough and at its end. Close observation of these pictures enables us to conceive of the importance of the apparently simple act of coughing and shows how justified is the medical practice that tends to diminish it in some bronchopulmonary processes.

It is important to call attention to the strangulation that occurs in the large bronchi at the end of coughing. This may be clearly seen in a partial record reproduced in Figure 16.

The modifications of the caliber of the bronchi during the act of coughing may be very irregular: some segments of the bronchus show no reduction in their anterior diameter, while in adjacent segments strangulation may be observed. In Figure 17 we see that during the act of coughing a general reduction of the tracheobronchial caliber has taken place, but in an irregular manner, with a semistrangulation of the inferior common stem. From this figure we may gather that there exists a functional obstacle which creates a hypertension in the segments ventilated by these bronchi.

The act of coughing seems to take place simultaneously in both lungs, but our bronchographic observations show that patients cough preferably with only one lung, as if the "irritative thorn" that produces the reflex led to a local and not a general response. In Figure 19 we see what happened in a boy during inspiration and coughing. In these bronchograms it is evident that the cough has taken place in the left and not in the right lung. It can also be noticed that the esophagus has not been influenced by the cough.

We have stated that in the act of cough-

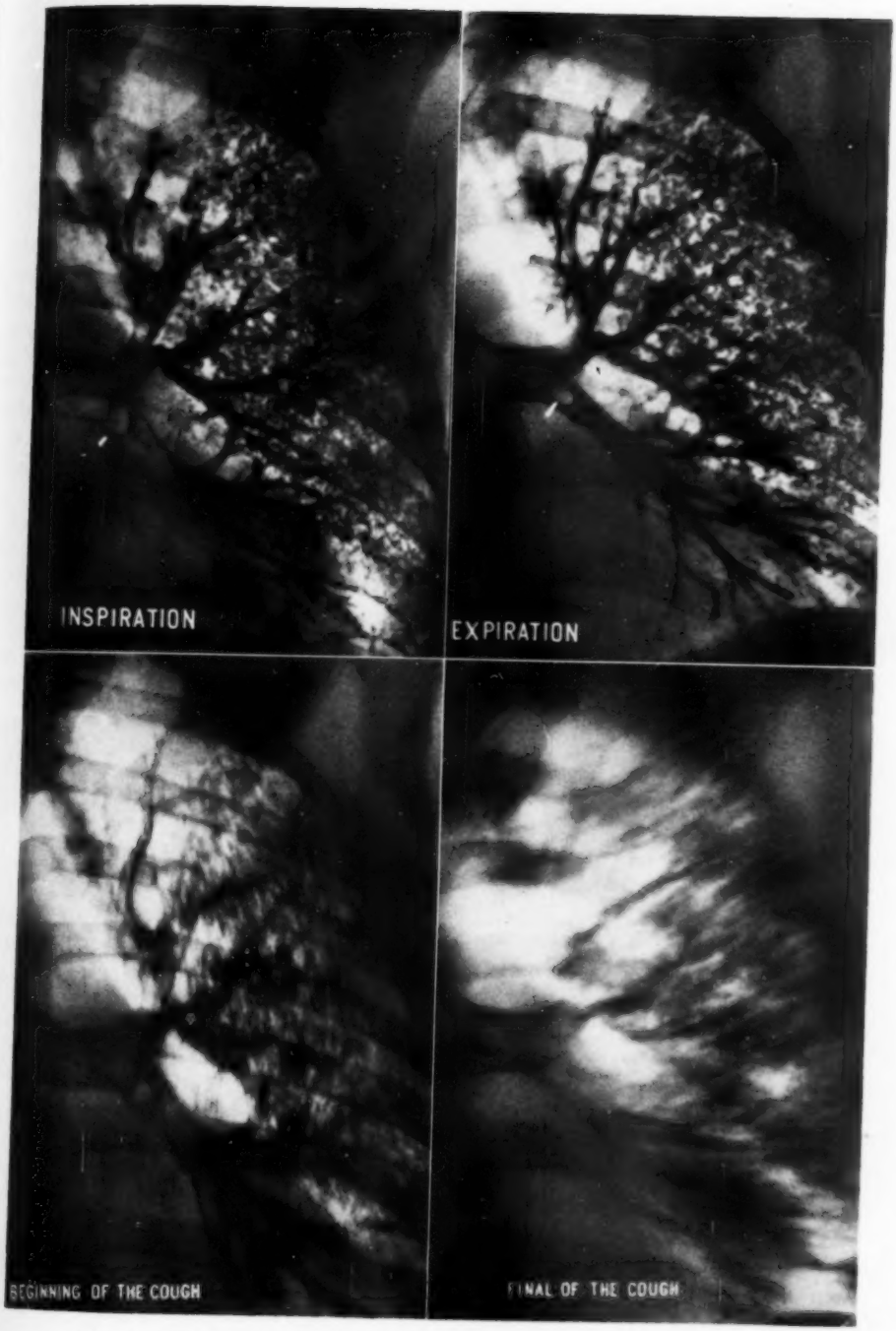


Fig. 15. Serial record of inspiration, expiration, beginning and end of coughing.

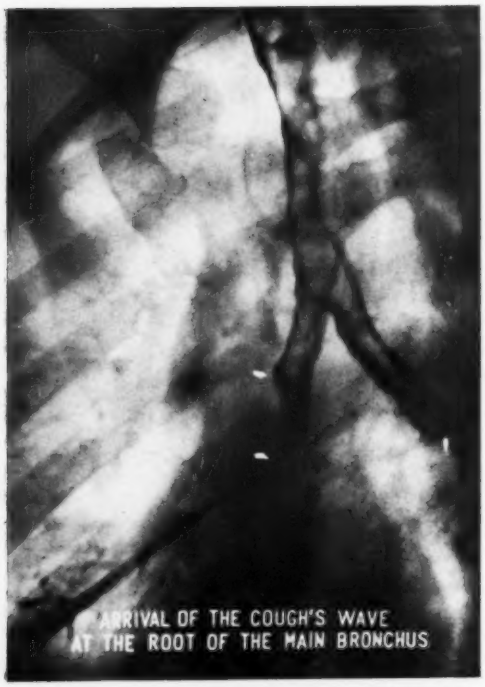


Fig. 16. End of cough. Notice strangulation of the main bronchi.

ing the bronchial wall contracts intensely in its subchondral part and that a contractive wave is produced which passes from the fine branching, where it starts, to the large trunks. It is an active ejaculation of the bronchial wall and not a simple thoracodiaphragmatic hypertensive phenomenon which, by reducing the space, expels the contents.

We see proof of this fact in observing what happens during cough in bronchiectasis. During this act, the ectasic portions which are associated with the destruction of the active lining of the wall retain the opaque contents, as seen in Figure 20. If destruction of the wall is general, that is if destruction has involved most of the wall, nothing can be evacuated, and only recumbency will favor expulsion of the contents. These dynamic characteristics permit the differentiating of reversible bronchiectasis, susceptible to medical treatment, from the irreversible disease, which must be submitted to early surgical treatment. Coughing permits the exact differentiation, for when the mucous mem-

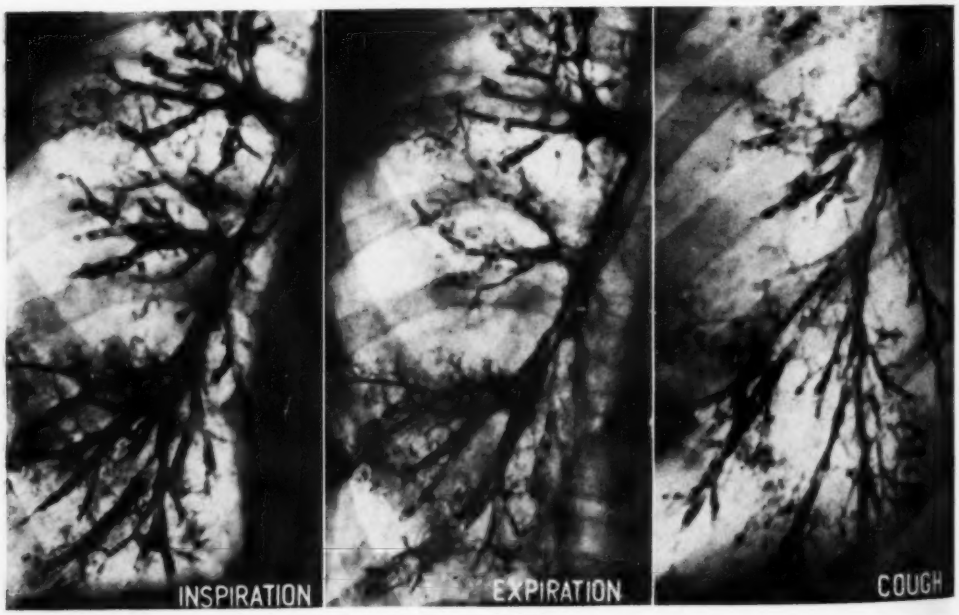


Fig. 17. Serial record of inspiration, expiration, and cough in a patient suffering from chronic bronchitis. Notice the truncular strangulations produced during expiration and also during cough; also the reduction of caliber (very irregular) produced in the branches of the different lobes.

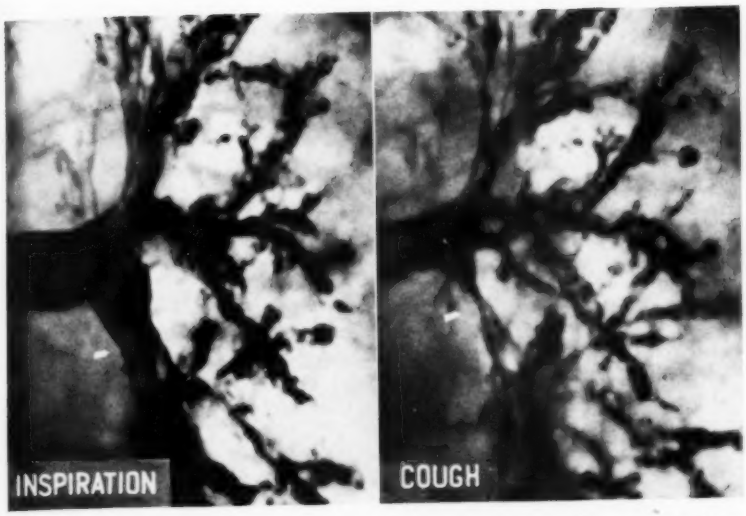


Fig. 18. Appearance at the instant of inspiration and during the act of coughing. Notice the semistrangulation of the main inferior bronchus, also the truncular strangulation.

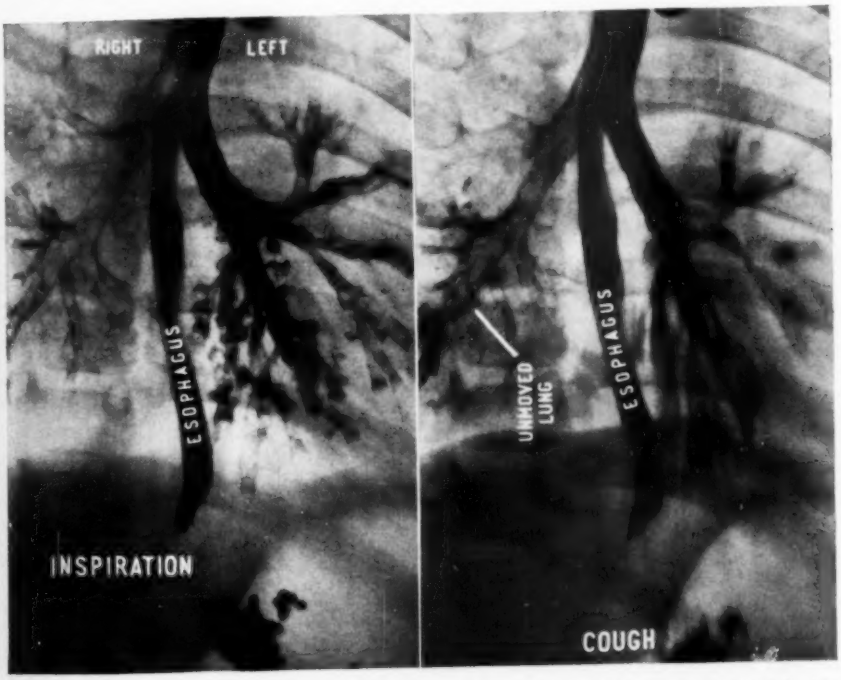


Fig. 19. Bronchograms taken during inspiration and coughing. Only one lung, the left, has been involved in the act of coughing.

brane of the bronchus is destroyed the opaque medium is not expelled.

Figure 21 illustrates one of these cases, with an initial bronchiectasis, in which retention of the opaque medium in some branches was observed during coughing.

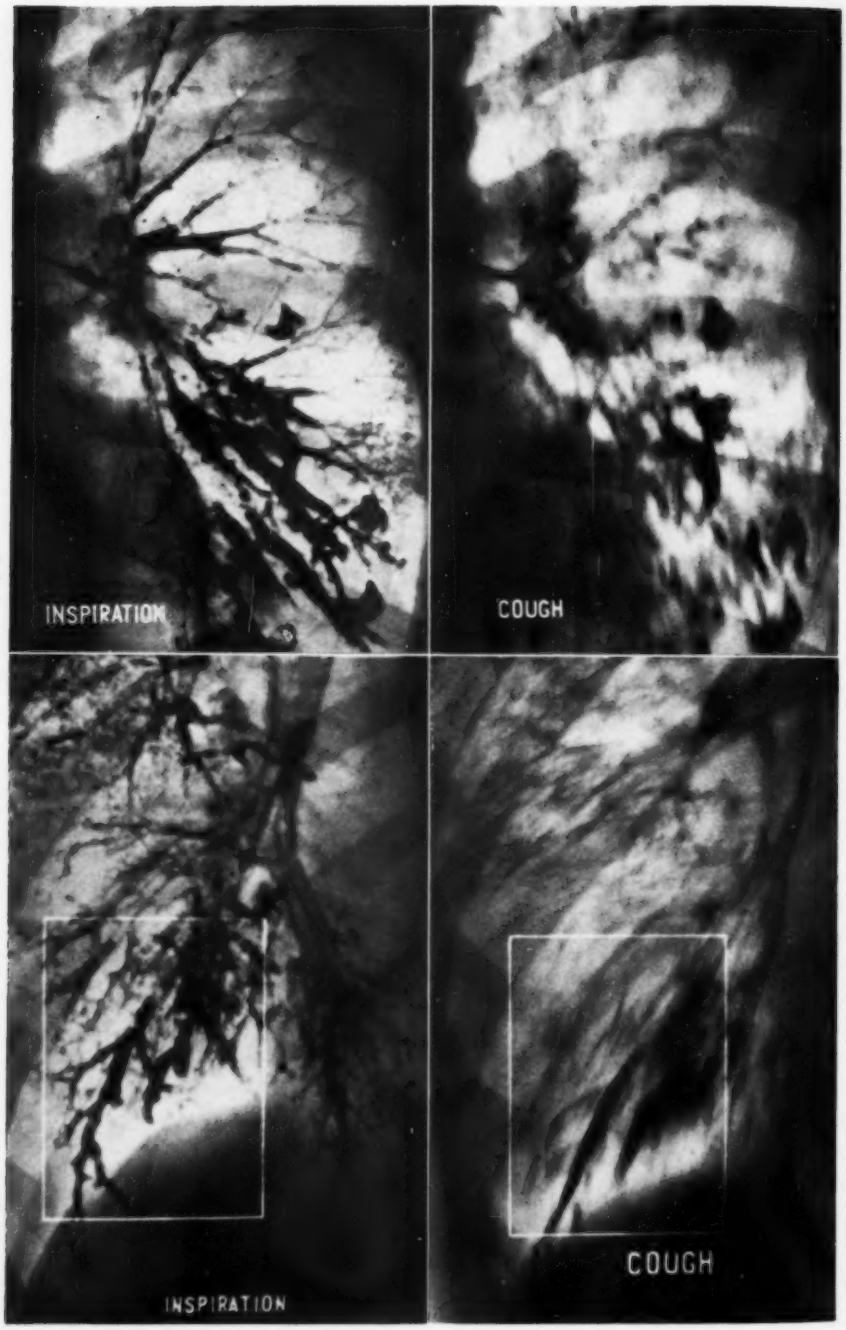


Fig. 20 (above). Effect of cough on the bronchial content, observed in a bronchiectatic patient. The contents are expelled only where no destuctive zones of the bronchial wall exist.
Fig. 21 (below). Slight bronchiectasis in the inferior lobe and same case during coughing. It is seen that there are some branches retaining the opaque medium.

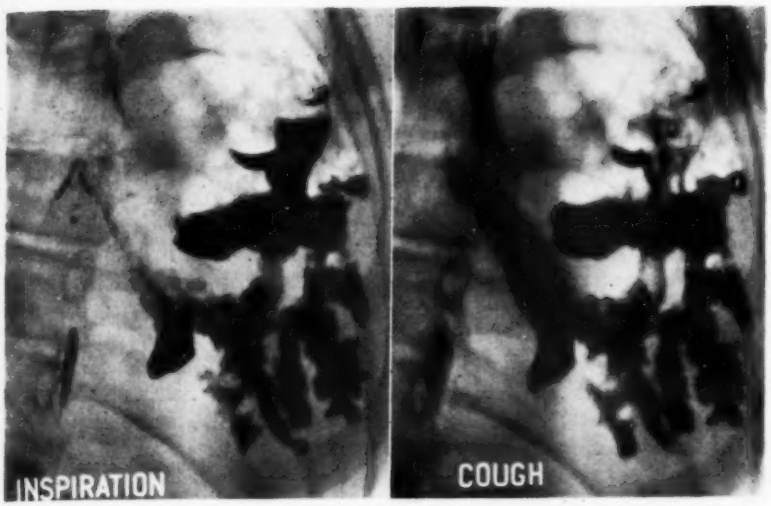


Fig. 22. Bronchograms showing that the expulsion of the bronchial contents is due to contraction of the bronchial wall. When the latter has been destroyed, there is no expulsion.

These are precisely the cases to be treated by surgical resection, as the anatomic conditions will never permit their medical cure.

SUMMARY

The caliber of the bronchus is increased during inspiration and reduced during expiration.

Regional modifications take place simultaneously at the roots of the bronchial branches, playing the role of functional sphincters. These are scarcely noticeable under normal conditions but are very manifest in the presence of irritation of the mucous membrane as, for instance, in bronchial asthma.

These variations of caliber and movement are of great importance in regulating the ventilation of the lungs.

During the act of coughing substantial changes take place in the form and size of the bronchi, chiefly of the primary and secondary branches, and including the trachea.

The bronchographic studies presented show that the act of coughing consists of a swift peristaltic wave which runs from the small bronchi toward the trachea. The thoracic and diaphragmatic pressure are secondary factors favoring but nor causing expulsion of the bronchial contents.

In confirmation of this theory, it is shown that during the cough the content of other thoracic organs, as the esophagus, is not mobilized, and that the act of coughing can be accomplished exclusively in a single lung or even in a single lobe.

It is also shown that in bronchiectasis the secretions are not expelled by coughing for, since the mucous and muscular sheet has been destroyed, the peristaltic wave cannot originate in the bronchiectatic branches.

These dynamic characteristics make possible a differentiation between reversible and irreversible bronchiectasis.

Av. General Paz 151
Córdoba, Argentina

(For Spanish Summary see following page)

SUMARIO

Dinamismo Bronquial

El calibre del bronquio aumenta durante la inspiración y disminuye durante la expiración.

Tienen lugar simultáneamente modificaciones regionales en las raíces de las ramas bronquiales, desempeñando así el papel de esfínteres funcionales, que apenas distinguibles en condiciones normales se vuelven muy manifiestos en presencia de irritación de la mucosa, como, por ejemplo, en el asma bronquial. Esas variaciones de calibre y movilidad revisten mucha importancia en la regulación de la ventilación pulmonar.

Durante el acto de toser ocurren alteraciones sustanciales en la forma y el tamaño de los bronquios, principalmente en las ramas primarias y secundarias, y comprendiendo la tráquea. Los estudios broncográficos muestran que el acto de la tos consiste en una veloz onda peristáltica que va de los bronquios pequeños hacia la tráquea. La presión torácica y la diafragmática son factores secundarios que favorecen, pero no ocasionan, la expulsión del contenido bronquial. Confirmando esta teoría, demuéstrase que durante la tos no se moviliza el contenido de otros órganos torácicos, como el esófago, y que el acto de toser puede completarse exclusivamente en un solo pulmón y hasta en un solo lóbulo.

Demuéstrase también que, en la bronquiectasia, no se expulsan las secreciones tosiendo, porque, destruídas las capas mucosa y muscular, la onda peristáltica no puede tener su punto de origen en las ramas bronquiectáticas. Esas características dinámicas permiten hacer la diferenciación entre la bronquiectasia reversible y la irreversible.

DISCUSSION

(Papers by Rottenberg and Golden; Di Rienzo)

Leo G. Rigler, M.D. (Minneapolis, Minn.): I want first to discuss the excellent review of the etiology of spontaneous pneumothorax presented by Dr. Golden and Dr. Rottenberg. They confirm by factual evidence the impression which most of us with any experience in this field have had, that most of such cases outside of the tuberculous sanatoriums are non-tuberculous.

My own experience with spontaneous pneumothorax was with a series of cases, reported some years ago, in apparently healthy young adults, curiously enough almost all males, attending the University, who would come in after a sudden attack of pain and dyspnea. Exhaustive studies never revealed any evidence of other abnormalities in this series, which numbered 11 cases in two years. As a matter of fact, we see such cases among the large university population all the time. We have one medical student who has had four separate attacks, and yet we have never been able to demonstrate any underlying pathological process.

I should emphasize that some of these pneumothoraces are very small in size and may easily be missed if studies are not made in different positions. It is particularly important to get films in both expiration and inspiration, because a small pneumothorax will become quite easily apparent during expiration when it is almost invisible, or at least very difficult to see, during inspiration.

I think that Dr. Di Rienzo's paper is one of the most important and one of the most beautiful demonstrations that we have had before this Society in a long time. He has really given us a visual demonstration of the phenomena of respiration and of the cough. It is astounding to be able to see the act of coughing, as it were, on the screen. I'm lost in admiration of his beautiful and brilliant technic.

Some years ago, we tried to do something like this, but we were unsuccessful. We did succeed in demonstrating to our satisfaction the changes in the length and caliber of the bronchi which occur during inspiration and expiration and a few other phenomena of this kind.

I believe that Hans Jarre, with his cineroentgenographic unit, some years ago was able to show some of these phenomena, but I think that Dr. Di Rienzo has demonstrated them to a much greater degree.

I would like to emphasize particularly some of the points which he has made, especially regarding the loss of elasticity of the bronchus, its destruction, and his demonstration of the lack of respiration in segments of the lung in which either occlusion or destruction of the bronchi has oc-

curred. The matter of bronchial spasm is of a somewhat different character and requires a little further study.

Some years ago, we were interested in studying cases of bronchial asthma and were impressed with the important role which mucus in the bronchi plays in these matters which have been largely attributed to spasm. In an allergic bronchial asthma, I am confident (and this has been confirmed by work that we did on animals) that, in the later stages, bronchial spasm is not nearly so important a factor in the development of emphysema as is the accumulation of mucus in great quantities within the bronchus. The occlusions that we see are due, at least in considerable part, to the formation of mucous plugs. This can be very well shown in careful cross sections of the bronchi made in the removed lung. Some of the findings which Dr. Di Rienzo showed illustrate very beautifully, I think, the coiling up of the mucous membrane which occurs as the bronchus contracts. The mucous membrane seems to become redundant, and thus tends to reduce the size of the bronchial lumen. I was particularly impressed with his brilliant demonstration of the mucous membrane pattern, as a result either of this coiling up or of actual inflammatory processes.

I should call your attention to some work that Anderson Hilding of Duluth did on the tracheobronchial tree in chickens. He demonstrated quite effectively that the mucus passes up the bronchi in the form of little piston-like tubes which seem to carry air with them. This mechanism is a factor at least in the removal of air from segments of the lung which have become obstructed, even though the circulation also acts to remove air.

I would like to discuss Dr. Di Rienzo's paper at much greater length, because it contains so much food for thought. I think, however, that it would be quite impossible to do this in the time available. I would certainly recommend that everyone look at his exhibit and study it in detail. I would also recommend that everyone read his paper in detail, because it contains so much important information which can clarify some of these phemomena that we see exhibited.

Dr. Felix G. Fleischner of Boston, Masssachusetts, was also to be here to discuss Dr. Di Rienzo's paper, but he is unable to be present because of illness. He sent me a written discussion which I should like to read, because it also contributes to our knowledge of this subject.

Felix G. Fleischner, M.D. (Boston, Mass.): Dr. Di Rienzo has for many years pursued bronchographic studies of a particular type. Though the visualization of the bronchial tree is commonly used to establish morphological changes such as bronchiectasis, bronchial stenosis, or tumors, he has presented observations on the normal and abnormal physiology of the bronchi.

It has been known that the bronchi widen and lengthen during inspiratory expansion of the lung, and become narrower and shorter during its expiratory collapse. However, this has never before been demonstrated by such excellent documentary roentgenograms.

Dr. Di Rienzo has also demonstrated the rhythm and speed by which the opaque oil is drawn into the smaller branches of the bronchial tree and has suggested the significance of disturbances of this rhythm. We may go a step further. From bronchospirometric studies we have learned that some part of the lung may partake in the ventilation to a lesser degree than another, though it may look perfectly well aerated on the roentgenogram. Studying the rate of inflow of the oil by fluoroscopy or repeated roentgenograms taken at short intervals—using this technic as a kind of refined bronchospirometry—we may see that the oil does not penetrate into the peripheral branches of a certain lobe, segment, or subsegment, even in the absence of gross obstruction. Such a lobe, though apparently well *aerated* on the roentgenogram, takes no part or participates only incompletely in the play of *ventilation*, the respiratory air exchange. This lobe may be rigid due to interstitial fibrosis or emphysema, or arrested due to pleural adhesions and partial thoracic immobilization. Its air filling is more of a stationary type, with just enough air oozing into the peripheral portions to be continuously resorbed by the alveolar capillaries. There is very little if any ventilation, and such a lobe may be worthless for the function of respiration. It is apparent that information of this kind may be valuable in the planning of thoracic surgery in addition to the data obtained by vital capacity studies, bronchospirometry, determination of oxygen absorption, etc. Certainly such information gives us useful insight into the functional capacity of the lung.

I am not so convinced of the validity of all Dr. Di Rienzo's theoretical deductions. The bronchi have a muscular system and it is obvious that these muscles have a function. Their spiral arrangement ("geodesic" arrangement, according to Miller) has been thoroughly explored; their rhythmic contractions and relaxations have been demonstrated through the recording of their action currents by means of electrobronchography by Luisada and others. These recorded waves are synchronous with the normal respiration. The spastic narrowing of bronchi in bronchial asthma and related conditions is generally accepted as a fact. However, the lengthening and shortening as as well as the widening and narrowing of the bronchi during forced respiration can be largely explained by mechanical stress and strain exerted on the bronchial wall by the surrounding parenchyma. The irregularity of the caliber and con-

tour of the narrow bronchi, and particularly those indentations projecting into the bronchial lumen, so prominent on several of the author's slides, suggest a sphincteric action of circular muscle bundles. However, if we examine specimens of pulmonary lobes in a stage of collapse, as recovered at surgery or autopsy, we occasionally find circular mucosal folds in bronchi of the third, fourth, or fifth order. The pathologist explains these folds as due to heaping up of the movable mucosa when the bronchi shorten and narrow down with the collapsing lung. This heaping up of the mucosa contributes considerably, in the living as well, to the narrowing of the bronchial lumen when the bronchus as a whole becomes shorter and narrower. And I believe that it is more this heaping-up effect of the mucosa than muscular contraction that causes the spurs and ring-like indentations in most of the author's admirable post-tussic bronchograms.

However, what the final answer to this academic controversy will be is not too important. Most of us have used bronchography as a means to study gross abnormalities of morphological kind in the bronchial tree. We are accustomed to look at a bronchogram as a still picture. Dr. Di Rienzo has shown us how to use bronchography as a tool for the study of normal and abnormal physiology of the bronchi and lung. We are grateful to him for his pioneer work.

Correlation Between the Roentgenologic and Pathologic Findings in Chronic Pneumonitis of the Cholesterol Type[1]

LAURENCE L. ROBBINS, M.D., and RONALD C. SNIFFEN, M.D.

Boston, Mass.

THERE HAS BEEN seen with increasing frequency during the past few years a form of chronic pneumonitis that hitherto we have not recognized in the absence of bronchial obstruction. The process characterized by its chronicity, with either acute or insidious onset, has been considered as a particular type of chronic pneumonitis because of the unusual tissue reactions. The lung is involved in a chronic interstitial inflammation in which the exudate is largely composed of mononuclear cells filled with cholesterol and cholesterol esters. Often cholesterol is concentrated to such a high degree in the parenchyma that grossly the tissue appears bright yellow. This type of pneumonitis is common in the presence of bronchial obstruction and is often found in small localized areas in such chronic affections as bronchiectasis, pulmonary abscess, and tuberculosis. In the cases to be reported, however, no major bronchial obstruction or significant coexistent lung disease could be demonstrated roentgenologically or anatomically in the area of pneumonitis.

Why this condition is now appearing is difficult to explain. No description of it has been found in the literature other than Adams' (1) presentation of a group of patients with chronic, non-specific suppurative pneumonitis. The possibility that it might represent atypical healing of an infarct has been suggested, but nothing in the histologic studies has confirmed this supposition, even though cholesterol is frequently laid down in slow necrosis of tissue. That the process might be a variation of the ordinary lipoid pneumonitis has also been considered, but again certain factors, from the standpoint of history and of pathology, are not in accord with such a diagnosis. The lesion has been observed in cases in which neither sulfonamides nor penicillin has been administered.

The recognition of this type of pneumonitis may be in part due to the recent advances in thoracic surgery and anesthesia. With improvement in technic and the decreasing mortality rate, more and more conditions formerly considered inoperable are now being subjected to prompt surgical measures, particularly when there is the least suspicion of tumor.

This study is based on 11 cases seen at the Massachusetts General Hospital during the past four years. All but one of the patients were males; the only female was a child of twelve. The age range for the remainder of the group was from thirty-two to sixty-seven years. In more than half, the onset of illness was quite abrupt, characterized by pain, cough, fever, and sputum. In the others, the onset was insidious, with gradual development of cough, sputum, night sweats, weight loss, and in certain instances pain in the chest.

The sputum usually varied from mucoid to brownish; in only 3 cases was there frank hemoptysis. As a rule only bacteria of the species generally present in the respiratory tract could be isolated. Six of the patients, during the course of illness, had been treated with sulfa drugs or penicillin; only one had used nose drops containing oil. The duration of symptoms from onset to the time of operation varied from one and a half months to five years.[2]

---

[1] From the Departments of Radiology and of Pathology, Massachusetts General Hospital, Boston 14, Mass. Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

[2] Detailed description of clinical findings and treatment of this group of patients is being reported by Waddell, Sniffen and Sweet (18).

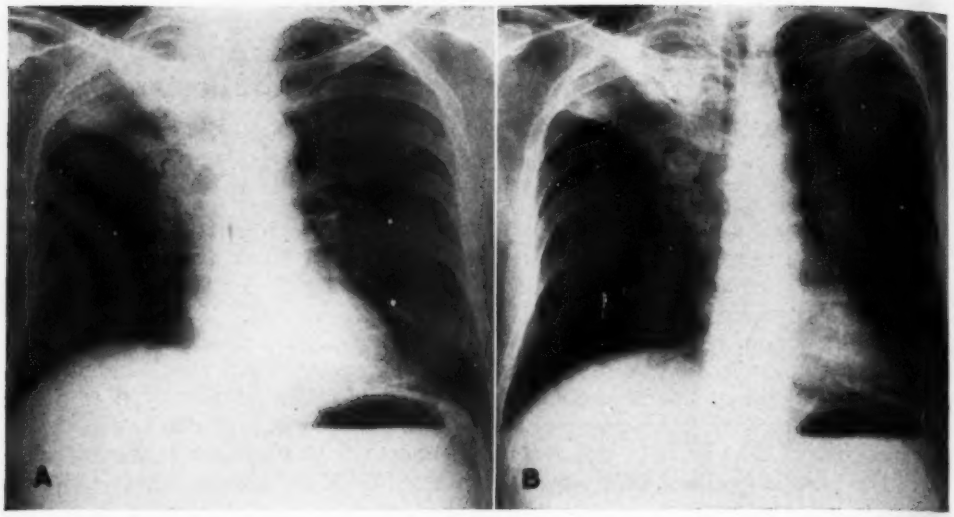


Fig. 1. Case 1: Male, 57 years. History of insidious onset one year prior to admission; cough productive of considerable sputum for two months; hemoptysis on three occasions during the month before entry; weight loss of 15 pounds; night sweats and low-grade fever at times. White blood count, 16,400. *Operation:* Right upper lobectomy. *Histology:* Chronic pneumonitis, chlolesterol type.

B. Grid film demonstrating partial collapse of the right upper lobe with evidence of enlarged nodes at the right hilus. The visualized bronchi are only questionably dilated.

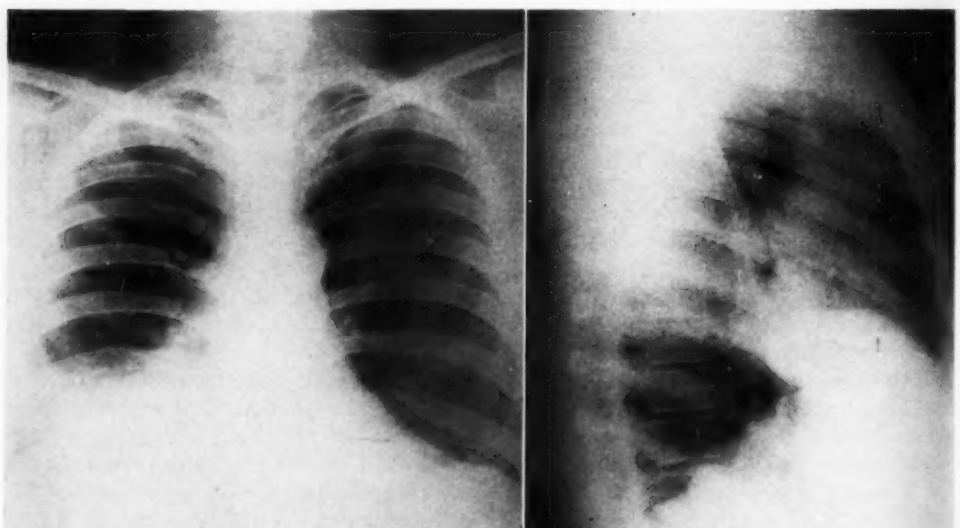


Fig. 2. Case 2: Male, 59 years. Ten months before admission the patient had a severe paroxysmal cough without fever, lasting for approximately one month. He was then well until three months before admission, when he experienced fatigability, dyspnea, and fever up to 102°. Penicillin produced some improvement, but dyspnea and fever continued. Pleuritic pain was present for five days prior to admission. Bronchoscopy was negative. *Operation:* Right middle lobectomy. *Histology:* Chronic pneumonitis, cholesterol type, of the medial segment. A small oat-cell carcinoma was found in the lateral segment.

A. Note the homogeneous density in the right lower lung field, as well as the enlarged nodes at the hilus.

B. Lateral view. The right middle lobe seems smaller than usual and homogeneously dense except at the apex.

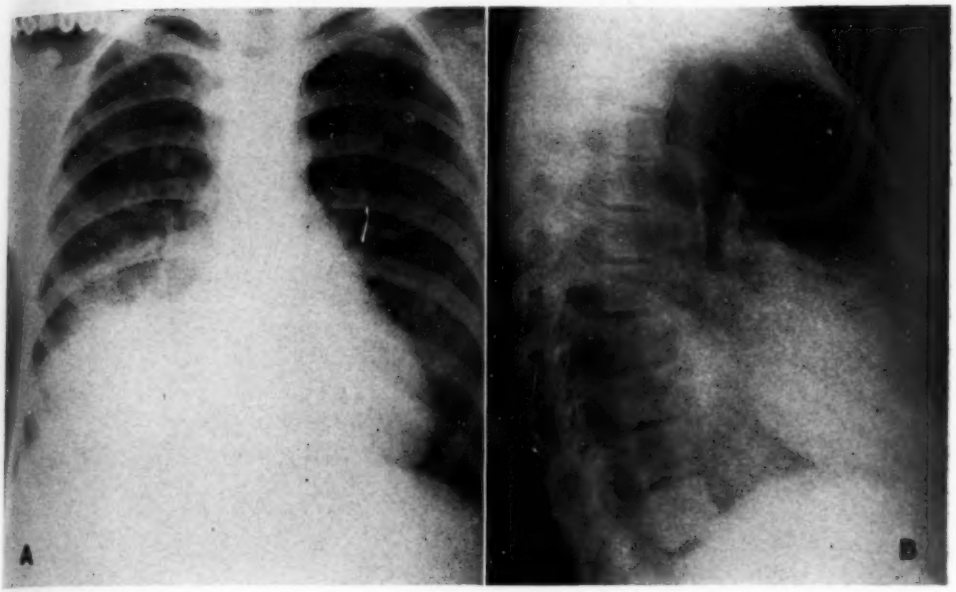


Fig. 3. Case 3: Female, 12 years. Ten months prior to admission there was gradual onset of cough, which became progressively more severe and productive of mucoid sputum. Six months later the cough was much worse and the patient noticed increasing fatigue. The white blood count ran as high as 20,000; temperature was seldom over 100°. Bronchoscopy showed chronic bronchitis. *Operation:* Right pneumonectomy. *Histology:* Chronic pneumonitis, cholesterol type.

A. Note the density which involves the greater portion of the right middle lobe, the anterior portion of the right lower lobe, and to some extent the right upper lobe.

B. Lateral view, confirming location of process and showing that there is no typical segmental involvement.

Treatment of these patients has been surgical for two reasons: often the lesion could not be differentiated from tumor, and, even when the diagnosis was made preoperatively, the few cases observed over a period of time had shown extension rather than healing of the process.

ROENTGENOLOGIC APPEARANCE

In the majority of instances, this type of pneumonitis has been confused roentgenologically with tumor, lung abscess, or infarct. After a few cases in which the lesion was considered an entity, or a special type of chronic pneumonitis, by the pathologists, the roentgenologists attempted to determine certain features that would be of help in preoperative diagnosis. It has been found in the series reported here that the roentgen appearance of the process is of two types. In 5 cases, there was rather extensive involvement of a lobe, whereas in the other 6 the lesion was localized to a portion of one or more segments. In neither group was there any correlation between the roentgenologic type and the type of symptomatic onset.

In that group of cases in which there was involvement of the greater portion of a lobe, it was noted that a moderate amount of collapse was present, although extreme degrees were not observed. The usual signs of collapse were readily apparent. There was a tendency for the shadow to be of a rather homogeneous density, conforming in shape to the portion of the lobe which it occupied. Variations in density, such as perhaps might be expected from the deposits of cholesterol within the fibrous tissue, were not observed. In those few cases in which the bronchi were visualized, they did not appear to be particularly dilated. (Figs. 1–3.)

In the second group, the area of increased density involved only a part of one or more segments of a lobe. Although the

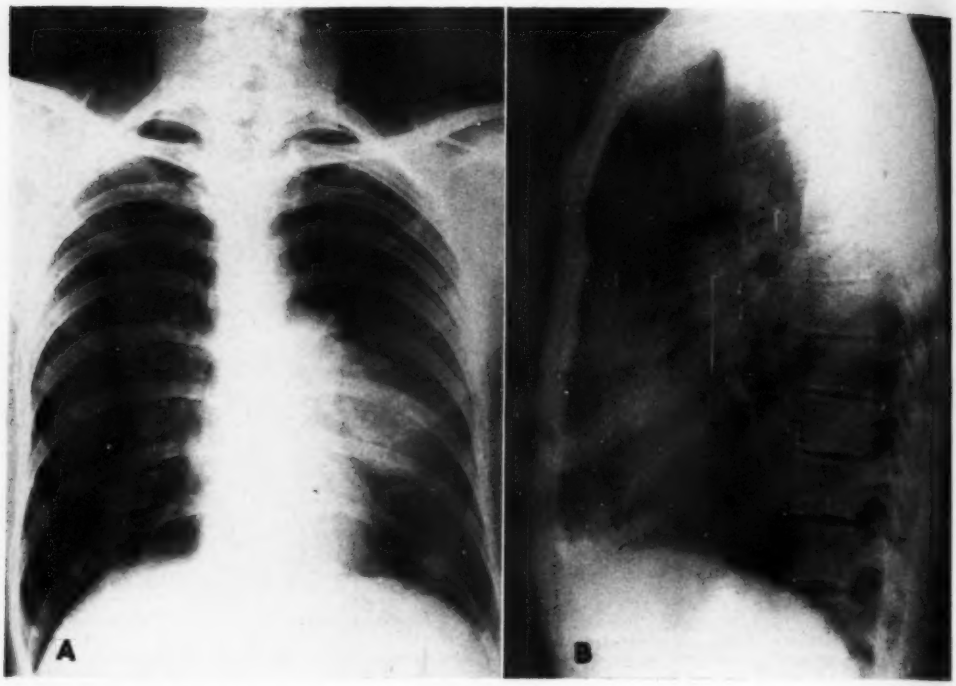


Fig. 4. Case 4: Male, 32 years. Nine months before admission, the patient noticed a feeling of pressure in the left anterior chest. Five months later cough developed, which was usually unproductive. It became progressively worse, and the patient experienced increasing night sweats. There was a loss of 10 pounds in weight. The temperature was usually normal, occasionally rising to 101°. The white count was 13,100. *Operation:* Left upper lobectomy. *Histology:* Chronic pneumonitis, cholesterol type.

A. Note the homogeneous density which obscures the left border of the heart.

B. Lateral view. The density is confined to the anterior portion of the lingula, and the posterior margin is seen to be sharply defined and slightly lobulated.

segment was at times somewhat smaller than normal, it was not invariably involved in its entirety. The shadow was seen to lie against the pleura, either peripherally or along a fissure, the long dimension being parallel with the pleural surface. The margin away from the pleura was rounded or lobulated and sharply defined. (To demonstrate this margin adequately, it may be necessary to make a careful fluoroscopic study as well as to take several films in various projections; otherwise the margin will be rather ill-defined.) As a rule, the bronchi were not visualized, but when they were, they appeared normal or only slightly dilated. (Figs. 4–8.)

In both types, widespread and localized, there was occasionally evidence of pleural reaction, either in the form of thickening or of pleural fluid. Enlargement of the hilar and mediastinal lymph nodes was sometimes apparent, and in rare instances small cavities, not over 1 to 1.5 cm. in diameter, were observed; they did not seem to be typical of lung abscess or of bronchiectatic cavities.

ROENTGENOLOGIC DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of chronic pneumonitis of the cholesterol type, the most common alternatives to be considered are tumor, lung abscess, and infarct.

The diagnostic points suggestive of *tumor* are (*a*) reduction in the size of a lobe or segment of a lobe; (*b*) a shadow suggestive in density and configuration of a tumor mass; (*c*) enlargement of the hilar and mediastinal lymph nodes.

(*a*) Collapse of a lobe due to extensive

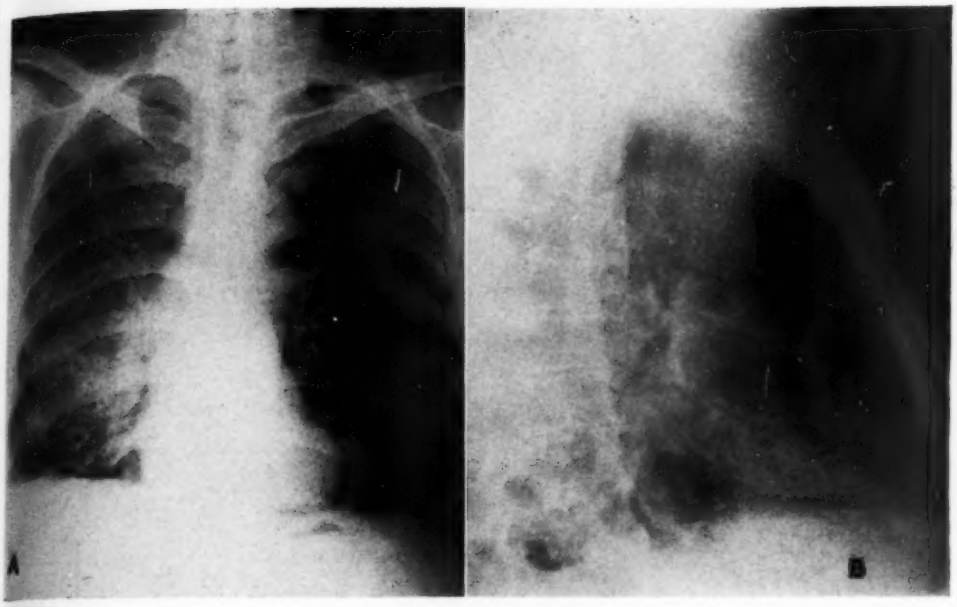


Fig. 5. Case 5: Male, 32 years. Eight months before admission the patient became unconscious and was hospitalized for "virus pneumonia." There were some fever and dark sputum but no chest pain. Cough and sputum persisted without evidence of hemoptysis. The white blood count ranged as high as 15,000. Bronchoscopy was negative. *Operation:* Right lower lobectomy. *Histology:* Chronic pneumonitis, cholesterol type.
A. Note the ill-defined density just below the right hilus. Within it are areas of rarefaction suggestive of slightly dilated bronchi. There is evidence of pleuritic reaction at the costophrenic angle.
B. Lateral view. The right lower lobe is seen to be partially collapsed, and posteriorly there are shadows of density against the pleura, with rounded, sharply defined anterior margins.

inflammatory involvement and that caused by tumor will be difficult or impossible to differentiate unless an actual intrabronchial mass is demonstrated.

(*b*) The shadow of increased density in pneumonitis is ordinarily less sharply defined than that due to the presence of a primary peripheral tumor or metastatic lesion. In certain instances the margins are somewhat irregular and lobulated, but the lobulations may be larger than those seen in a peripheral tumor. The fact that usually only a portion of a segment is involved in the inflammatory process is of help in differentiating pneumonitis from a tumor obstructing a smaller bronchus. In those cases in which iodized oil has been introduced, it has been found that several small bronchi may come to an abrupt stop, indicating that the degree of collapse present is not the result of obstruction of a single large bronchus.

(*c*) Enlarged hilar and mediastinal lymph nodes differ in no way from enlarged nodes due to tumor.

The rather discrete shadow of increased density may suggest the possibility of *lung abscess* before cavitation has occurred. When a cavity is present, its size is a factor of importance in distinguishing cholesterol pneumonitis from lung abscess; in the former, an occasional small cavity, seldom larger than 1 to 1.5 cm. in diameter and standing out less clearly than in abscess, is a characteristic finding. In abscess, the cavity as a rule develops at a much earlier stage of the patient's illness, and the adjacent pneumonitis is less marked. The cavities of cholesterol pneumonitis, visualized roentgenologically, are more suggestive of those seen in association with bronchiectasis or obstructive pneumonitis than of primary lung abscess. This is true not only because of their size but because of the faintness of their demonstration, the latter being due prob-

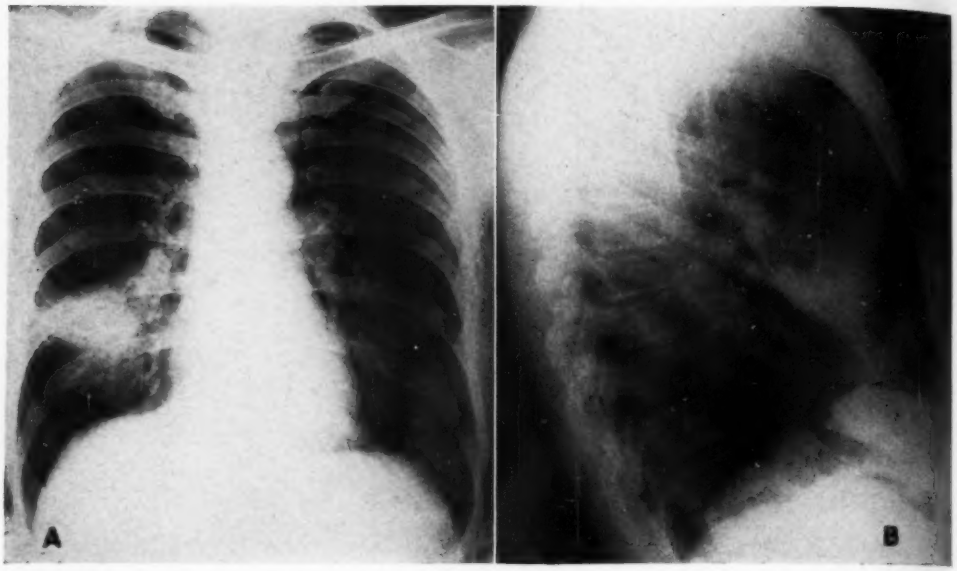


Fig. 6. Case 6: Male, 56 years. Four weeks before admission the patient had a severe head cold, followed in two weeks by cough, productive of rusty foul-smelling sputum. The cough persisted; the sputum became white except on two occasions, when it was blood-streaked. The white blood count was 7,900. Bronchoscopy was negative. *Operation:* Right middle lobectomy. *Histology:* Chronic pneumonitis, cholesterol type.

A. Note the area of density in the right lower lung field. The shadow is sharply defined and contains a few areas of rarefaction, suggesting small cavities.

B. Lateral view. The density is seen to be somewhat lobulated, lying in the mid portion of the middle lobe, extending from one fissure to the other.

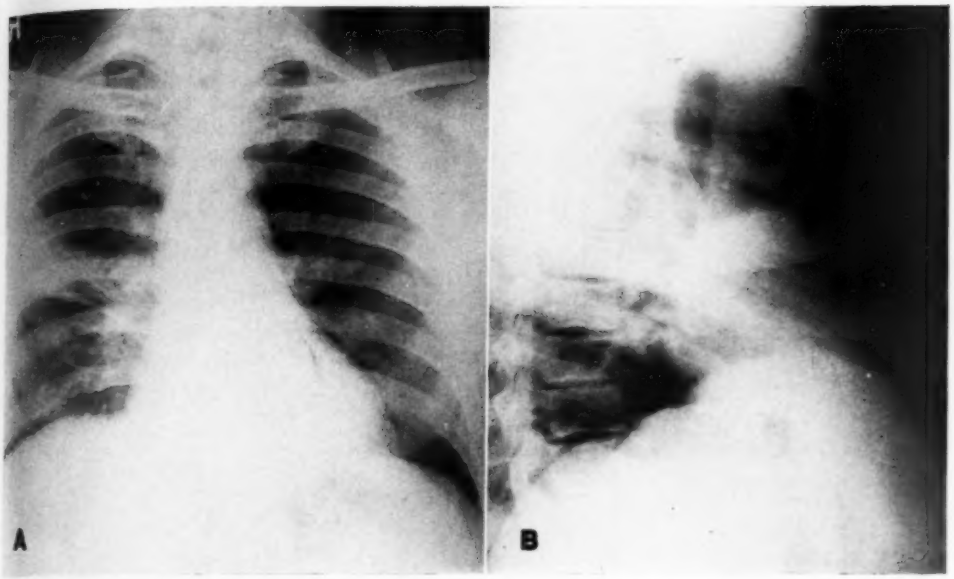
ably to the extensive surrounding areas of inflammation.

In many cases the shadow caused by cholesterol pneumonitis is similar to that of *pulmonary infarction* (4). The facts that in both conditions the shadow lies against the pleural surface, and that the long dimension is parallel to this surface, are quite confusing. In pneumonitis, however, the location is not so commonly at the junction of two pleural surfaces as in pulmonary infarction, and the margins are more sharply defined. If observation over a period of time is possible, it will be seen that in pneumonitis the process tends to spread slowly, whereas an infarct usually heals characteristically in a relatively short time. In some cases of pneumonitis a temporary decrease in the size of the area of density may later be followed by an increase, but at no time in the late stages is the shape or appearance of a healing infarct suggested. The sharp definition and the slight lobulation that may be present in the pneumonic shadow would be unusual findings in pulmonary infarction.

Pneumonitis due to aspiration of foods or mineral oils is, as a rule, a more diffuse process than the cholesterol type and is seen particularly in the lower lobes. The areas of density are less sharply defined and are frequently multiple, in contrast to the single area more typical of cholesterol pneumonitis.

PATHOLOGY

As no patient in our series came to operation in less than a month and a half after the onset of the pneumonia, the earliest and undoubtedly the most revealing phases of the inflammatory process were not encountered. The anatomic changes were essentially similar in all the specimens; the differences lay merely in the stage of healing attained by the tissue at the time of removal. The appearance of the lung could not be anticipated from the apparent duration of symptoms, for sometimes ad-



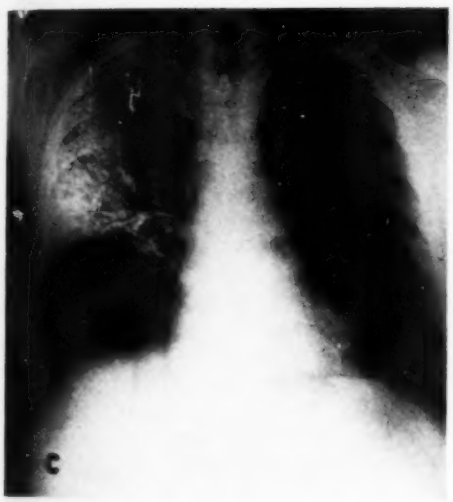


Fig. 7. Case 7: Male, 45 years. Nine weeks before entry the patient experienced sudden generalized aching, with fever and an increase in his chronic, unproductive cough. A course of penicillin resulted in some improvement, but the cough persisted and became productive of about one-half cup of sputum, occasionally blood-streaked. There was some pleuritic pain in the right anterior chest. The patient had lost 20 pounds in weight. Bronchoscopy was negative. The white blood count was 8,300. *Operation:* Right upper and middle lobectomy. *Histology:* Chronic pneumonitis, cholesterol type.

A. Note the area of density in the lateral portion of the base of the right upper lobe. This appears to be homogeneous in density.

B. Lateral view. The area of density lies against the septum, and its superior margin is sharply defined and rounded.

C. Bronchogram reveals slight, if any, dilatation of the bronchi to the area of involvement. Note the enlarged lymph nodes outlined at the right hilus.

vanced parenchymal scarring had taken place in a comparatively short time. The situation was further complicated in some instances by secondary bacterial invasion.

The affected lobe was always contracted in rough proportion to the amount of fibrosis. Fibrous adhesions involved all the pleural surfaces and were particularly dense at the site of the lesion. The bronchial lymph nodes, when included with the specimen, were remarkable for their size, often reaching a diameter of 2.0 cm.

The process involved either the major portion of the lobe or the whole lobe; in the former event, the diseased tissue fanned out from the hilus to assume a pyramidal shape with the base at the pleura. It did not adhere strictly to pulmonary segments. At least one pleural surface was always involved by the pneumonia, and at operation the interlobar fissures were often found to be obliterated (Fig. 9). The fissures were not penetrated except in one instance, in the youngest patient, where a middle lobe lesion had

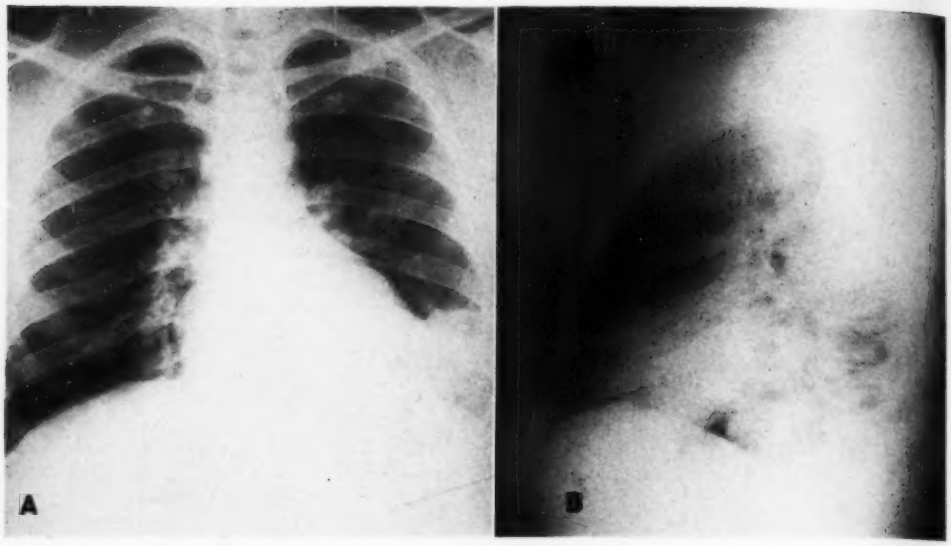


Fig. 8. Case 8: Male, 31 years, with history of sudden onset of pain in the left chest, becoming increasingly severe, unproductive cough, and fever of 100°. The patient had had similar episodes four and six years before, and had experienced a sense of pressure in the left chest every winter. The white blood count was 19,800. The cough persisted and became productive of non-odorous yellow sputum. *Operation:* Left lower lobectomy. *Histology:* Chronic pneumonitis, cholesterol type.

A. Note the sharply defined shadow in the left costophrenic angle, with rounded superior margin.

B. Lateral view. The shadow is seen to lie in the left lower lobe and has a somewhat lobulated margin.

A bronchogram showed no particular dilatation of the branches of the left lower lobe bronchus. The smaller bronchi terminated abruptly at the margin of the lesion.

spread to the adjacent tissue of the upper and lower lobes.

In the comparatively early phases of the process, the area of pneumonitis was an intense yellow, the result of close approximation of minute golden yellow dots. The primary lobules, although indistinct, were not obliterated, while the secondary lobules were sharply outlined by markedly thickened septa. As the healing process progressed, the parenchyma became gray and fibrous, while the yellow color faded and was localized to zones of less advanced carnification. Areas of emphysema were interspersed between the fibrous bands and involved the parenchyma of the lung adjacent to the pneumonitis. The border of the lesion, where it did not abut the pleura, was somewhat irregular and lobulated but clearly defined.

The larger bronchi of the diseased lobes were chronically inflamed and thickened. The smaller bronchi were occasionally acutely inflamed, dilated, and obviously destroyed, with their lumens filled by tenacious mucopurulent material. This change was not uniform throughout the diseased tissue and seemed to be caused by an acute reaction to recent bacterial invasion. In 4 cases the necrotizing bronchiolitis had led to the formation of a small abscess, never more than 1.5 cm. in diameter. Only borderline dilatation of the large bronchi was present in a small minority of the specimens.

On microscopic examination, the earliest alteration encountered in the parenchyma of the lungs was a massive influx into the air space of the primary lobule of large mononuclear cells with central or somewhat eccentric nuclei (Fig. 10). The cytoplasm of the majority of these cells, which often attained giant proportions and contained multiple nuclei, was composed of a foam of fine droplets. The infiltration was accompanied or followed closely by a chronic interstitial pneumonitis, beginning in the connective tissue of the septa and

periobronchial and perivascular regions, with subsequent involvement of the alveolar walls. The tissue response was characterized by edema and a lymphocyte and plasma-cell infiltration; the lymphocytes were frequently aggregated in follicles. These changes were almost entirely interstitial and played little part in the formation of the alveolar exudate. In addition, the alveolar septal cells became intensely swollen; they lined the entire primary lobule, and many of them seemed to be desquamating into the air spaces. It is probable that, by the acquisition of phagocytic powers after desquamation, these cells gave origin to the intra-alveolar foam cells.

The next remarkable step in the process was a gradual accumulation of vacuolated macrophages within the alveolar walls, coincident with a numerical decrease within the air space, implying that there was migration of the cells into the walls, although many of them may have been cleared from the air spaces by inclusion in the sputum. This resulted in marked swelling of the walls of the alveoli, so that the alveolar spaces were obliterated, and the primary lobules were consequently represented at this time merely by respiratory bronchioles and alveolar ducts (Fig. 11). A network of reticulum now appeared between the vacuolated cells in the alveolar walls, and as these cells decreased in number collagen was laid down (Fig. 12). In this manner the primary lobules were reduced to distorted respiratory bronchioles and alveolar ducts, separated and choked off by dense bands of collagen (Fig. 13).

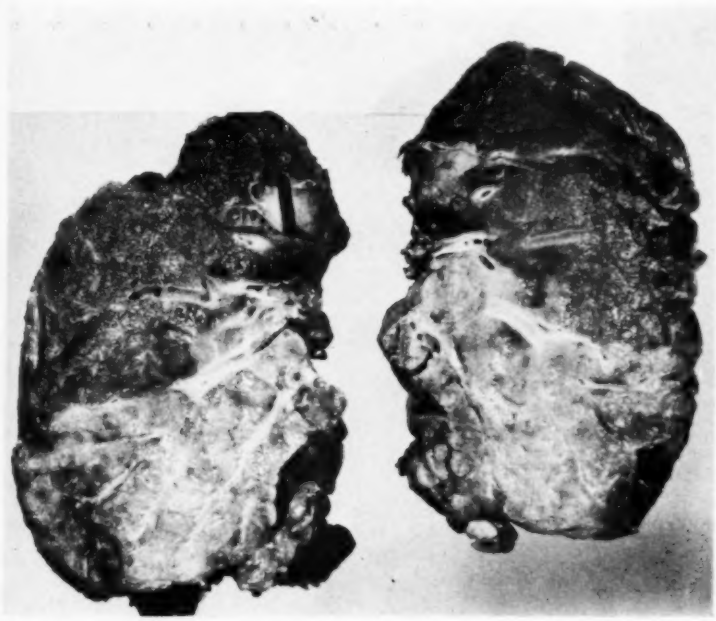


Fig. 9. Gross section of a right middle lobe, showing an early lesion in its upper half that was a homogeneous golden yellow color. The pneumonitis involves the lobe from hilus to pleural surface. Thickened septa outline the secondary lobules of the parenchyma. The margin which does not abut a pleural surface is irregular and quite sharply defined.

The ramifications of the large bronchi in the areas of pneumonitis usually showed an irregular chronic inflammatory infiltrate without significant destruction of the wall elements or dilatation; occasionally small mucosal ulcerations were present (Fig. 14). The bronchioles showed a similar change, but in a few cases an acute reaction was

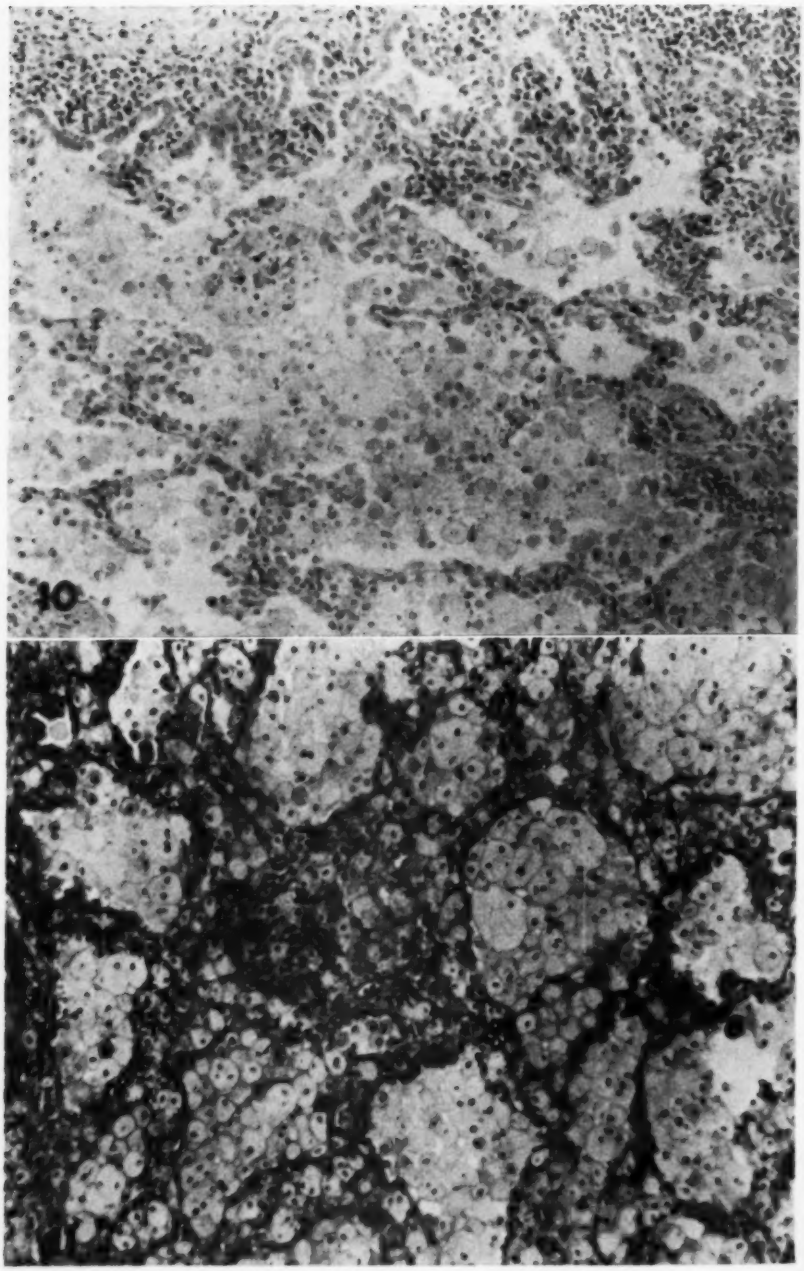


Fig. 10. Microscopic section of the earliest changes encountered in the cases studied. The air spaces are occupied by large foamy monocytes containing cholesterol. The septal cells are swollen and the alveolar walls contain lymphocytes and plasma cells. ×170

Fig. 11. Marked infiltration of the alveolar walls by foamy macrophages. This has resulted in obliteration of the alveoli. The monocytes lying in the air spaces occupy alveolar ducts. ×180

superimposed that resulted in a necrotizing bronchiolitis and dilatation of some of these structures. With the advent of fibrotic changes within the lobule, the terminal bronchioles were held in rigid dilatation.

During the phase in the process in which these studies were made, the initial stages of the disease seemed to have passed, and the tissues at this point appeared to be concerned with the disposition of a foreign substance. This substance lay within the endarteritic thickening and perivascular fibrosis in the later stages of the process. The lymphatics were unobstructed and did not contain vacuolated monocytes; nor were these cells present in the bronchopulmonary lymph nodes, which showed merely non-specific inflammatory changes. It is probable that the cholesterol is gradually absorbed from the alveolar walls as fibrosis increases. Indeed, small amounts of the substance were found in cells resembling fibroblasts, suggesting that

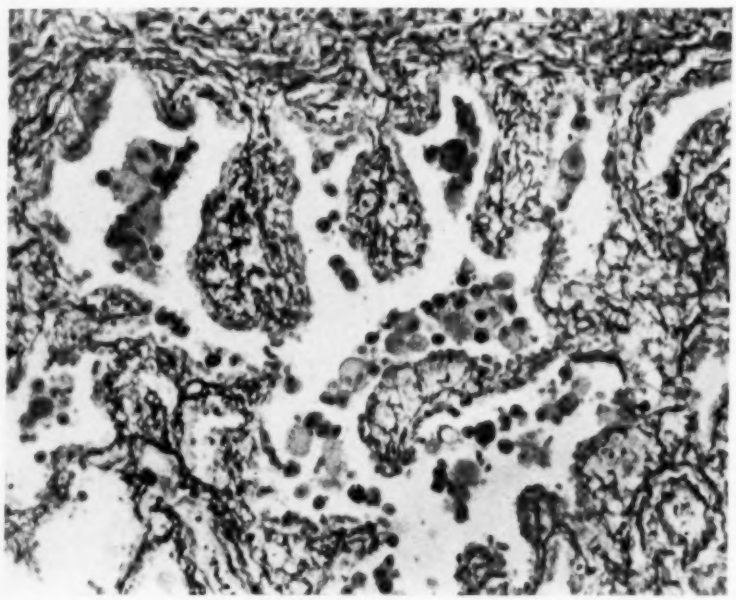


Fig. 12. Silver stain showing the fine reticulum fibrils deposited around the monocytes in the alveolar walls. No fibrils are present in the intra-alveolar exudate. ×160

large monocytes of the air spaces and alveolar walls in the form of fine intracytoplasmic vacuoles. It was intensely sudanophilic and doubly refractile (Fig. 15), was precipitated by digitonin in fine needles, and gave a positive Schultz reaction for cholesterol and cholesterol esters. Chemical analysis of grossly yellow areas in the lungs gave values for cholesterol and its esters as high as 24 and 90 times the normal, respectively.

The lymphatics and blood vessels throughout the areas of pneumonitis were normal, except that the latter showed the macrophages are transformed into fibrocytes.

The end-result of the process in cases which do not come to operation is not clear. At autopsy we have not seen similar pulmonary changes in the absence of associated lung disease. At the present time there is no reason to believe that this process goes on to abscess formation or bronchiectasis. The evidence at hand favors fibrosis as the final stage.

Our interest in this group of patients was stimulated by the absence of coexistent lung disease, particularly the absence of

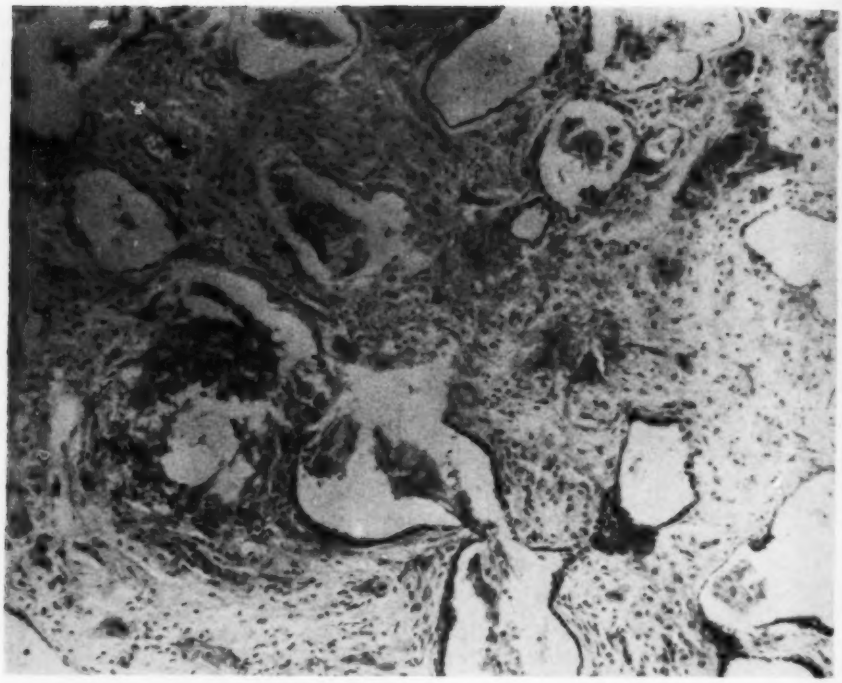


Fig. 13. Fibrosis of the thickened alveolar walls, leaving only distorted and choked alveolar ducts in the lobule. A terminal bronchiole is present, running upward from the lower margin. ×170

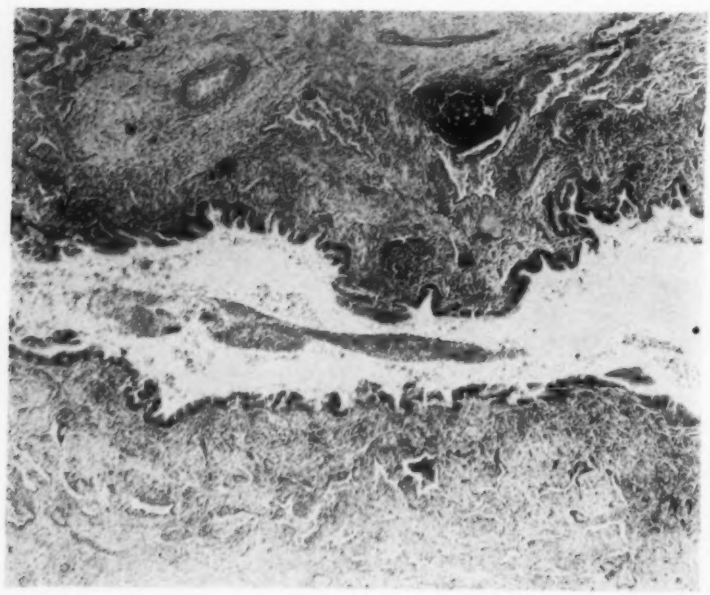


Fig. 14. A tertiary bronchus with chronic inflammatory changes in the wall. The lumen contains a polymorphonuclear exudate. ×35

bronchial stenosis. In one case a small oat-cell carcinoma was found at the hilus, but as this originated in a primary bronchus which did not supply the area of pneumonitis, it was thought that the tumor played no part in the production of the pneumonia. "Drowned lung" with a high cholesterol content and a similar tissue reaction is common in bronchial stenosis from whatever cause, but in these patients obstruction in major bronchi supplying the area of pneumonitis could not be demonstrated by any means. Microscopic sections, however, often showed a mucoid or mucopurulent exudate in the bronchioles. This exudate is a possible source of bronchial obstruction, and it might be perpetuated by a smoldering reaction in the lungs or by repeated acute exacerbations of pneumonitis following secondary infection.

In the stages of the process which were studied, the tissues seemed to be dealing with a foreign substance, though hampered in their efforts by mild secondary infection. Indeed, the similarity between this type of pneumonitis and aspiration pneumonia, due to paraffin oil, is striking. In paraffin-oil pneumonia, the substance is engulfed in large monocytes within the air space of the primary lobule. Later these cells migrate into the alveolar walls, where they excite a chronic interstitial pneumonitis and a reticulum and collagen response. There are, however, striking differences between the monocytes in the two conditions. Paraffin-oil droplets soon coalesce to form large globules in the cytoplasm, that push the nuclei to one side, and the oil is not doubly refractile. Furthermore, it dissolves the Sudan stains rather feebly and does not give the histochemical reactions of cholesterol.

The condition under discussion is not similar to an ordinary unresolved pneumonia, as there is no organization of the intra-alveolar exudate, and fibrin is absent except in the event of secondary infection. Unresolved pneumonia shows very few vacuolated monocytes containing a substance that is doubly refractile. Only

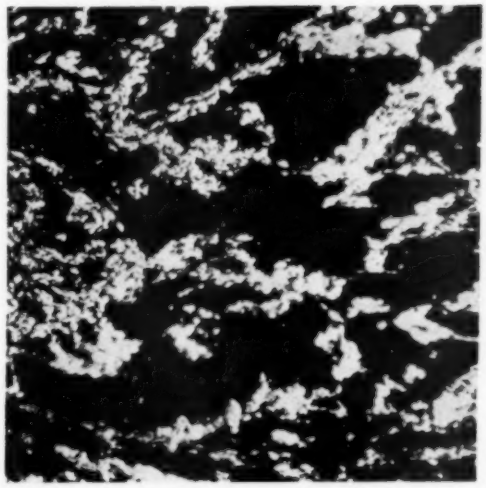


Fig. 15. Anisotropic material in the alveolar walls (precipitated by digitonin). ×170

half the patients in the group received sulfonamides and/or penicillin during the course of their illness. It is unreasonable, therefore, to implicate these forms of therapy in a modification of the histologic picture of a more common type of pneumonia. No bacteriologic studies were made of the resected lobes, and bacteria stains on tissue sections were not contributory.

The possibility that these lesions might represent the healing phase of complete or incomplete infarcts of the lung was considered. Histologically, however, the conditions are dissimilar, and when blood enters the alveolar spaces without necrosis of the alveolar walls, both clinically (4) and experimentally (17), it is cleared away very rapidly. No gross or microscopic vascular occlusions could be found in the specimens.

A background of chronic interstitial pneumonitis with a mononuclear response and swelling of the alveolar septal cells is present in primary atypical pneumonia (2, 6, 14). In reports on that disease, however, no mention is made of cholesterol deposition nor were such deposits described by Kay (5), who studied lobectomy specimens from men in whom bronchiectasis developed in the course of the pneu-

monia. There is an interstitial inflammation with large numbers of mononuclear cells and swelling of the septal cells in the pneumonias associated with certain of the viral (8, 10, 19) and rickettsial diseases (3, 7, 9), and possibly in rheumatic fever (15). A somewhat similar tissue response can be elicited by roentgen rays in man and animals (20, 21, 22), and by the intratracheal injection of dilute bacterial toxins (16) or vaccine virus (13) in animals. By injecting vaccine virus and bacteria simultaneously into the lungs of rabbits, McCordock and Muckenfuss (12) were able to reproduce the pulmonary changes and complications seen in patients dying during the course of influenza, measles, and whooping cough. In the reports on the pneumonias mentioned above, no reference could be found to monocytes filled with cholesterol. Hyaline membrane formation in the alveolar ducts is a feature of many of these pneumonias, a phenomenon that was not encountered in the lungs under discussion here. No inclusion bodies were found in the respiratory epithelium. MacCallum (11) in his description of the pneumonias associated with influenza remarks on the presence of many "fat-laden" monocytes in the lungs of three men dying from three to seven weeks after the onset of the disease.

Obviously a wide variety of agents can excite the same fundamental tissue response in the lungs, and the quality of the reaction is therefore of little help in determining the etiologic agent. It has been stressed in this report that only what appeared to be the late manifestations of the pneumonitis have been observed. The histologic evidence at hand would seem to indicate that all the changes recorded in this study can be explained on the basis of an attempt by the pulmonary tissues to dispose of precipitated cholesterol and its esters. These substances appear to be mildly irritating to the lung, like paraffin oil (an attempt is being made to determine this point experimentally). The intriguing problem to be unravelled is what set of conditions leads to the deposition of large amounts of cholesterol in the air spaces. The fact that bronchial stenosis produces the lesion fairly regularly may be a valuable clue, but the role of such stenosis is not immediately obvious. One can be reasonably certain that there was no obstruction in the major bronchi during the period of study, yet the destruction in the bronchioles and the plugging of their lumens by tenacious exudate were sometimes quite impressive. Obstruction in many bronchioles may well have the same total effect as stenosis of a major bronchus. For the time being the origin of the cholesterol is unknown. There is no convincing evidence that it is produced locally.

SUMMARY

Eleven cases of chronic pneumonitis, treated surgically, have been presented. The outstanding feature of the pneumonia in these cases is the deposition of cholesterol and cholesterol esters in the air spaces in the absence of coexistent lung disease.

Certain roentgenologic findings should at least suggest the diagnosis of chronic pneumonitis of the cholesterol type. It is fully realized that it will be impossible to exclude tumor in certain cases, but in those presenting the roentgen signs described above there should be a strong suspicion of the correct diagnosis.

Whether this type of chronic pneumonitis is a definite entity is questionable. The roentgenologic and pathologic evidence indicates that some obstruction of the smaller bronchi or bronchioles is often present, and this may be a determining factor in laying the background for the particular type of reaction that has been observed.

The end-result of the process is not clear, but we do not favor the hypothesis that it is a forerunner of abscess formation or bronchiectasis. The material at hand seems to indicate the subsequent development of fibrosis in the area of pneumonitis.

NOTE: We wish to acknowledge the efforts of Dr William Waddell in collecting this material.

REFERENCES

1. ADAMS, W. E.: Chronic Nonspecific Suppurative Pneumonitis. Surgery **22:** 723–724, 1947.
2. DINGLE, J. H., AND FINLAND, M.: Virus Pneumonias. Primary Atypical Pneumonias of Unknown Etiology. New England J. Med. **227:** 378–385, 1942.
3. FINLAND, M., AND DINGLE, J. H.: Virus Pneumonias. Pneumonias Associated with Known Non-Bacterial Agents: Influenza, Psittacosis and Q Fever. New England J. Med. **227:** 342–350, 1942.
4. HAMPTON, A. O., AND CASTLEMAN, B.: Correlation of Postmortem Chest Teleroentgenograms with Autopsy Findings, with Special Reference to Pulmonary Embolism and Infarction. Am. J. Roentgenol. **43:** 305–326, 1940.
5. KAY, E. B.: Bronchiectasis Following Atypical Pneumonia. Arch. Int. Med. **75:** 89–104, 1945.
6. KNEELAND, Y., JR., AND SMETANA, H. F.: Current Bronchopneumonia of Unusual Character and Undetermined Etiology. Bull. Johns Hopkins Hosp. **67:** 229–267, 1940.
7. LILLIE, R. D.: Pathology of Psittacosis in Man. U. S. Public Health Bull. **161:** 1–46, 1933.
8. LILLIE, R. D.: Smallpox and Vaccinia. The Pathologic Histology. Arch. Path. **10:** 241–291, 1930.
9. LILLIE, R. D., PERRIN, T. L., AND ARMSTRONG C.: An Institutional Outbreak of Pneumonitis. Histopathology in Man and Rhesus Monkeys in Pneumonitis Due to Virus of "Q" Fever. Pub. Health Rep. **56:** 149–155, 1941.
10. MACCALLUM, W. G.: Pathology of Pneumonia in U. S. Army Camps During Winter 1917–18. Johns Hopkins Hosp. Rep. **20:** 1–147, 1921.
11. MACCALLUM, W. G.: Pathological Anatomy of Pneumonia Associated with Influenza. Johns Hopkins Hosp. Rep. **20:** 149–249, 1921.
12. MCCORDOCK, H. A., AND MUCKENFUSS, R. S.: Similarity of Virus Pneumonia in Animals to Epidemic Influenza and Interstitial Bronchopneumonia in Man. Am. J. Path. **9:** 221–252, 1933.
13. MUCKENFUSS, R. S., MCCORDOCK, H. A., AND HARTER, J. S.: Study of Vaccine Virus Pneumonia in Rabbits. Am. J. Path. **8:** 63–72, 1932.
14. REIMANN, H. A.: Viral Pneumonias and Pneumonias of Probable Viral Origin. Medicine **26:** 167–219, 1947.
15. SELDIN, D. W., KAPLAN, H. S., AND BUNTING, H.: Rheumatic Pneumonia. Ann. Int. Med. **26:** 496–520, 1947.
16. SPRUNT, D. H., MARTIN, D. S., AND WILLIAMS, J. E.: Interstitial Bronchopneumonia. J. Exper. Med. **62:** 73–84, 1935.
17. STRASSMAN, G.: Formation of Hemosiderin in the Lungs. An Experimental Study. Arch. Path. **38:** 76–81, 1944.
18. WADDELL, W., SNIFFEN, R. C., AND SWEET, R. H.: Chronic Pneumonitis. Its Clinical and Pathologic Aspects. Report of 10 Cases Showing Chronic Interstitial Pneumonitis and Unusual Deposits of Cholesterol. To appear in J. Thoracic Surg., October, 1949.
19. WARING, J. J., NEUBUERGER, K. T., AND GEEVER, E. F.: Severe Forms of Chickenpox in Adults, with Autopsy Observations in a Case with Associated Pneumonia and Encephalitis. Arch. Int. Med. **69:** 384–408, 1942.
20. WARREN, S.: Effects of Radiation on Normal Tissues. V. Effects on Respiratory System. Arch Path. **34:** 917–931, 1942.
21. WARREN, S., AND GATES, O.: Radiation Pneumonitis. Experimental and Pathologic Observations. Arch. Path. **30:** 440–460, 1940.
22. WARREN, S., AND SPENCER, J.: Radiation Reaction in Lung. Am. J. Roentgenol. **43:** 682–701, 1940.

Massachusetts General Hospital
Boston 14, Mass.

SUMARIO

Los Hallazgos Radiológicos y Patológicos en la Neumonitis Crónica de Tipo Colesterol

Preséntanse 11 casos de neumonitis crónica, tratados quirúrgicamente, en los cuales la característica sobresaliente consistió en depósitos de colesterol y ésteres de colesterol en los espacios aéreos sin neumopatía coexistente.

Ciertos hallazgos roentgenológicos deben por lo menos indicar el diagnóstico de neumonitis crónica de tipo colesterol. El aspecto toma dos formas. En una hay invasión bastante extensa de un lóbulo, y en esos casos existe colapso moderado y la sombra es de un espesor bastante homogéneo conforme a la porción del lóbulo que ocupa. En los otros casos, la zona de mayor densidad comprende únicamente parte de uno o más segmentos de un lóbulo, y en ellos la sombra parece quedar contra una cara pleural, ya periféricamente o a lo largo de una fisura, quedando la dimensión más larga paralela a la cara pleural, en tanto que el borde alejado de la pleura es redondeado o lobulado y bien definido. Ni en uno ni en otro grupo observóse mayor dilatación bronquial. Es manifiesto que en ciertos casos resulta imposible excluir la presencia de tumor, pero en los que presenten los precitados signos roentgenológicos hay motivo poderoso para sospechar el diagnóstico acertado.

Parece dudoso que esta forma de neumonitis crónica constituya una entidad bien definida. Los datos radiológicos y patológicos indican que a menudo hay alguna obstrucción de los bronquios más pequeños o los bronquiolos, lo cual puede ser el factor determinante en el establecimiento de la base para la clase de reacción observada.

El resultado terminal del proceso no es claro. Los datos a mano parecen indicar la aparición subsiguiente de fibrosis en la zona de la neumonitis.

DISCUSSION

George T. Leclercq, M.D. (San Francisco): In the *Proceedings of the Staff Meetings of the Mayo Clinic* for Jan. 10, 1945, Drs. O. T. Clagett and J. R. McDonald present a case of bronchiectasis and lipoid pneumonitis with a large aberrant pulmonary artery, which is similar to the cases just presented. They state: "The fourth condition in which fat is present in the lung is that in which a purulent infection causes the breakdown of cells in the lung which releases fat which was intracellular. Thus fat which was invisible in cells becomes visible extracellularly and this fat is then picked up by the macrophages. This is commonly observed in suppurative disease throughout the body. . . . It appears likely that in the lung under discussion, the bronchiectasis represents the primary disease and that lipoid pneumonitis is secondary to the breakdown of cells which has resulted from the suppuration in the bronchiectasis. This is supported by the doubly refractile nature of the fat in the lung. . . . Chemical analysis of the involved portion of the lobe showed 4.1 per cent total lipoid as calculated by wet weight. There were 2.9 per cent cholesterol, 1.4 per cent cholesteral ester, 5.3 per cent lecithin, and 1.2 per cent fatty acid."

We have had no experience locally with this disease, but I should like to describe briefly one case we have recently observed.

A 53-year-old white male was transferred from a mental hospital for possible surgery because of a mass in the right lung with partial collapse of the right middle lobe, discovered on a routine film. Sputum examinations revealed what were thought to be malignant cells. A lobectomy was performed. Grossly the right middle lobe was pale yellow in color. Microscopic sections showed essentially what Dr. Robbins' cases revealed. They differed in that some of the alveoli were filled with fat and some with macrophages containing fat cells, while the picture was otherwise one of chronic pneumonitis. Chemical analysis showed 0.25 per cent cholesterol. It was presumed that what was observed was neutral fat and fatty acids.

We should like to know whether Dr. Robbins has encountered any such case.

Aubrey O. Hampton, M.D. (Washington, D. C.): I recall that Dr. Sniffen at one time (and I was greatly honored) appeared in our X-Ray Seminar to close a discussion by presenting the pathological findings. He discovered that the discussion didn't close then, and I believe that he was quite disappointed. His attitude has gradually changed, however, until now he has reached the point where he is about as pessimistic as we are, at least when he views something histologically. He is making no attempt, I am sure, to close the discussion in this case.

I had the unfortunate experience recently of completely overlooking a fulminating case of lipoid pneumonia, and that stimulated me to look up something on the subject. A case of unilateral lobular disease which I had seen in 1942 at the Walter Reed Hospital was called by the pathologist lipoid pneumonia, and I was quite disturbed over the diagnosis; in fact, I didn't believe it. Fortunately, the slide and the specimen were on file at the General Medical Museum, and we recovered them and had them studied completely, except unfortunately for chemistry. The histology of this lesion, so far as we can tell, was exactly the same as cholesterol pneumonia. It was yellow, and in all other respects it was the same. We had films of this patient before the development of the lesion, and we had follow-up films showing a recurrence on the opposite side, with localized areas of consolidation similar to infarcts, tumor, etc. Therefore, I'm anxious that we maintain the attitude that this is perhaps lipoid pneumonia of a localized type in spite of the fact that so far lipoid pneumonia has not been bright yellow.

I should like to mention some facts as to the incidence of lipoid pneumonia. I found the incidence in series of postmortem studies, up to 3,000, to be from 2 to 7 per cent, which shocked me greatly. I also found by casual inquiry that in all drugstores, practically, lethal nose drops were available which were being used quite casually, and that there is no Food and Drug Act which prevents their sale. I intend to discover, if possible, the present incidence of this disease in Washington, since we now have available all of the positive films from a survey of some 560,000 cases.

Dr. Robbins (*closing*): We have seen no case similar to Dr. Leclercq's. I should like to stress briefly the point that he brought out: that is, that the process occurs in bronchiectasis. We have seen many instances of cholesterol pneumonitis associated with bronchiectasis. I had an opportunity to talk with Dr. McDonald in September, and I asked him at that time if he had seen this type of pneumonitis in the absence of bronchial obstruction or bronchiectasis. He said that he had not.

As to the point made by Dr. Hampton, that this process might be one of the varieties of the lipoid group, we agree that cholesterol pneumonitis should be included in the group of lipoids, but we do not think it has any similarity, from the chemical standpoint, to the condition with which we are familiar as ordinary lipoid pneumonia.

I should like to ask all of you for help in this matter, because actually we do not know a great deal about it.

Active Bronchopulmonary Lithiasis[1]

EUGENE FREEDMAN, M.D., and JAMES H. BILLINGS, M.D.

Los Angeles, Calif.

LUNG STONES attracted the curiosity of physicians of ancient Greece and Rome, and according to Morgagni (quoted by Atlee, 2), pneumoliths were known to Areteus, Galen, and Aristotle. As autopsies became part of medical investigation, more knowledge was gathered about this condition. The earliest comprehensive clinical report was that of Schenck (39) in 1600 (Barrett, 5). In 1744 Boerhaave described the case of the famous botanist Sebastian Veillantius, who expectorated 400 stones (Stivelman, 42).

Leroy (24) in 1868 and Poulalion (32) in 1891 published the first accurate and modern discussions on the subject, and changed the name *phthysis calculosa* to *pseudophthysis calculosa*, appreciating the fact that pulmonary tuberculosis did not play an active part in the clinical syndrome of bronchopulmonary lithiasis. Other terms employed for the condition in modern medical literature are pulmoliths, pneumoliths, lung stones, and bronchial calculi. Lyter (26) designated as active bronchopulmonary lithiasis those cases in which calcifications cause clinical symptoms, and we believe this to be the most descriptive of all terms.

A complete review of the English and American literature since 1900 reveals a total of 96 recorded cases. The largest individual group was reported by Tinney and Moersch (43), who in 1944 collected 28 cases from the files of the Mayo Clinic. Graham, Singer and Ballon (18) observed 13 cases, Fox and Clerf (15) 10 cases, Myers (29) 7 cases, Vinson and Bumpus (45) 7 cases. The remaining 31 were mostly single case reports (1, 2, 5, 7, 8, 9, 12, 13, 14, 22, 23, 25, 26, 27, 28, 29, 31, 33, 35, 36, 41, 42, 44, 49).

In spite of the relatively meager literature on the subject, bronchopulmonary lithiasis is apparently more frequent than the available publications would indicate. In the X-Ray Department of a relatively small hospital of 310 beds, we have had the opportunity during the past year to study 7 proved cases.

PATHOLOGY

According to Poulalion (32) broncholiths may originate from the pleura, the lung parenchyma, the tracheobronchial lymph nodes, and from within the bronchi. The deposits usually develop in areas of previous inflammation or necrosis. Suppuration, hemorrhages, infarcts, fungous infections and pneumoconioses are known to leave calcific scars within the lungs, but pulmonary tuberculosis is by far their most frequent source.

Conditions producing hypercalcemia, such as hyperparathyroidism or extensive bone destruction due to various etiological factors, may lead to metastatic calcium deposits within the lungs (4, 10). Gander (16) and Harbitz (19) described cases of extensive alveolar wall calcification and ossification in the lungs, without finding any primary causative condition. Miliary calcium and bone deposits have been found in the lungs of patients suffering from mitral stenosis, probably originating in areas of hemorrhage (37, 38).

When forming within the bronchi, broncholiths may originate from anthracotic or silicotic material (22, 42), inspissated secretions, fibrin plugs, soft-tissue sequestra, or foreign bodies. Salivary calculi, rhinoliths, tonsilliths (31), or spicules of bone loosened during surgery on the sinuses or nose, may be aspirated and become broncholiths. Necrosis in the lung may lead to discharge of dead cartilage

[1] From the Department of Radiology, Cedars of Lebanon Hospital, Los Angeles, Calif. Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

fragments into the bronchi. The overwhelming majority of broncholiths, however, develop by perforation of calcific pulmonary foci or calcified lymph nodes into the tracheobronchial tree, instead of being formed primarily in the bronchi.

Most broncholiths are from 2 to 20 mm. in diameter and are grayish or brownish-white in color. Their surfaces may be smooth, irregular, or mammillated, and they are occasionally enclosed in a fibrous capsule. They may be solid or laminated and may even contain a liquid center (12). All analyses of lung stones agree in the reported proportion of their main constituents, namely, 10–15 per cent calcium carbonate and 85–90 per cent calcium phosphate. Wells (48) has pointed out that this proportion must be the same as in the bone, since the blood, in balance with the skeleton, maintains a fairly constant concentration level of these ions anywhere in the body. Brown (7), carefully analyzing several pneumoliths, found in addition to calcium carbonate and phosphate, traces of magnesium, oxalate, carbon, silica, iron, cholesterin, fats, mucus, and other organic material. Virchow's observations and Wells' (48) theory, supported by experimental evidence obtained by Tanaka and Hofmeister, that calcium deposits occur where a loss of acid raises the *p*H of the circulating fluids, are substantiated by the frequent finding of calcium in the lungs and in the kidneys, where carbonic acid and acid phosphates are given off. Calcifications within the stomach, which due to the loss of hydrochloric acid should be another theoretical site of calcium deposition, have been reported (4, 11) and are not uncommon in animals (17).

Wells (47) also demonstrated the similarity between pathological calcifications and physiological ossification. Not infrequently, broncholiths are found to contain areas of organized bone with haversian canals.

The stones may reach the bronchial lumen in either of two ways. According to Nager (30), an infection with suppuration may develop in the area of the calcific focus, causing mobilization and slow perforation through the bronchial wall of an adjacent and previously "silent" stone. The second type of mechanism through which stones can enter a bronchus has been described by Auerbach (3). According to him, the inspiratory expansion and the expiratory collapse of the bronchi and surrounding vascular structures may expose a certain section of the bronchial wall to the eroding action of an adjacent parenchymal or mediastinal calcification until a large enough gap is created for the calcification to enter the bronchial lumen.

Although the perforating calculi are usually traceable to old tuberculous nodes, it is remarkable that active tuberculosis is rarely accompanied by the expectoration of stones. Stivelman (42) found only 1 broncholith in 5,000 cases, and Pritchard (34) only 2 broncholiths in 7,000 autopsied cases of active pulmonary tuberculosis.

During the process of perforation the involved bronchial wall is the site of ulceration surrounded by granulating inflammatory tissue. Hemorrhage may occur from an exposed blood vessel in the ulcerated area or from the direct erosion by the stone of a large pulmonary vessel in the hilar region.

Partial or complete obstruction of a large or small bronchus may occur, depending on the size and shape of the calculus lodged within it. Atelectasis, pneumonitis, bronchiectasis, and abscess formation usually follow. Pleural effusion, empyema, pneumothorax, or mediastinal emphysema may develop. Histologic examination of the bronchial mucosa frequently shows squamous-cell metaplasia and increase in the number and activity of the goblet cells near the site of the broncholith.

CLINICAL SIGNS AND SYMPTOMS

The clinical picture may vary from complete absence of signs or symptoms to those of a severe or critical illness. The clinical manifestations are manifold and depend greatly on the degree of obstruction and the extent of the secondary inflam-

matory changes distal to the obstruction. Cough is usually paroxysmal, first dry, then productive. Pain is frequently localized in the parasternal area. It can vary from a mild soreness to a sharp "tearing" sensation. The paroxysm of cough and pain can be quite violent ("bronchial colic"). Hemoptysis is frequent, particularly after expulsion of the stone. The bleeding is usually slight, but in some instances is massive enough to necessitate transfusions and even an emergency lobectomy or pneumonectomy (6). Fatal hemorrhages due to bronchopulmonary lithiasis have been reported (6, 15). Fever, chills, leukocytosis and anemia are present, depending on the degree of the secondary inflammatory changes and the blood loss. Wheezing and dyspnea due to an obstructing stone and spasm from bronchial irritation can be severe and have been termed "stone asthma."

Bronchorrhea is caused by the increased goblet cell activity in the irritated mucosa. There are almost always weight loss and malaise during the periods when the patient is in active distress. During the expulsion phase, acute air hunger may develop in those cases where the stone is of such size as to become lodged in the larynx, unable to pass the vocal cords. Instrumental extraction of the stone may become necessary in such instances.

DIAGNOSIS AND ROENTGEN FEATURES

It is evident from the foregoing that the clinical picture varies to such an extent as to simulate practically every pathological condition which may develop within the chest. Before the correct diagnosis is established, the patient is usually thought to have one of the following conditions: chronic bronchitis, bronchiectasis, tuberculosis, lung abscess, chronic pneumonitis, pneumoconiosis, foreign body, pulmonary infarction, whooping cough, spirochete infection of the lungs (50), angina, congestive heart failure, and bronchial adenoma or carcinoma.

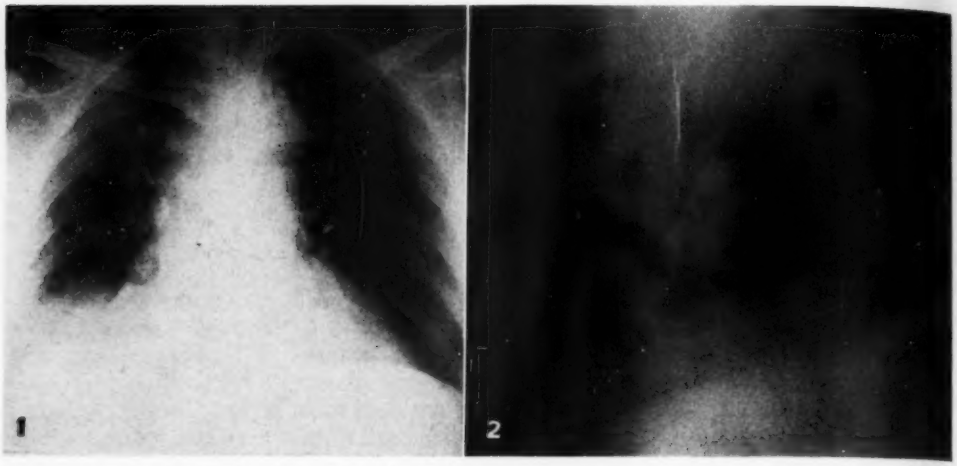
The physical examination is of little help in diagnosis. Bronchoscopy has become the most important diagnostic aid (20). It may reveal the broncholiths within the bronchi or arouse suspicion of a perforating calculus by revealing an ulcerating or granulomatous lesion in the bronchial wall. However, even in the presence of a negative biopsy, carcinoma is the condition which is most commonly suspected.

Vinson and Bumpus (45), who advocate bronchoscopy in every case of non-tuberculous pulmonary disease, report the finding of broncholiths in 7 unsuspected cases. Careful history taking, including direct questions with regard to concretions in the sputum, may lead to a correct diagnosis. Even more conclusive is the actual finding of the stones in the sputum. However, frequently the diagnosis is established only after the study of surgical or postmortem specimens.

As in many other conditions, a conclusive diagnosis is not possible on the basis of the roentgenologic findings. The evidence obtained, however, may be sufficient to arouse a strong suspicion of bronchopulmonary lithiasis, provided the radiologist is fully aware of this clinical syndrome. Hilar and perihilar calcifications in the chest film are common and are usually considered of little or no clinical importance. For this reason, many broncholiths are missed. In the presence of shadows suggesting carcinoma of the bronchus, atelectasis, lung abscess, or bronchiectasis, peripheral to calcific deposits, or with areas of calcification in their central portions, the radiologist should think of the possibility of active bronchopulmonary lithiasis and suggest this diagnosis to the clinician. In triangular areas of atelectasis, the calculus is frequently situated at the apex of the triangle. Perforating hilar node calcifications often lead to slight effusion in the interlobar fissures.

Follow-up roentgenograms of patients who have coughed up stones will show a decrease in the size or a disappearance of the calcification, associated with clearing of the atelectasis.

Laminagraphy is an important diagnos-



Figs. 1 and 2. Case 1. Anteroposterior and lateral views showing areas of calcification in and around the hilus and mottling in the right lower lung.

tic procedure, as it may show the stone lying in the lumen of a bronchus or demonstrate the calculus in close relation to the bronchial wall, with the secondary inflammatory changes peripheral to the point of obstruction. Bronchography is of decidedly less value than laminagraphy, but will occasionally demonstrate a point of bronchial obstruction.

TREATMENT

Spontaneous expulsion of the stone or stones takes place frequently, with characteristic relief of distress and symptoms in most instances (28). The literature contains several reports on successful removal of broncholiths by bronchoscopy (1, 15, 20, 22, 43, 45, 46). This, however, is not without certain risks. In severing the fibrous pedicles from the bronchial wall and extracting the stones, pneumothorax, mediastinal emphysema, and hemorrhages, even fatal, may ensue (1). Bronchial dilatation may be helpful in facilitating the eventual expulsion of the stone. In those instances where bleeding cannot be controlled, lobectomy or pneumonectomy may be required (1, 6). In general, the plan of treatment must be based on the type of pulmonary and vascular complications secondary to bronchial obstruction, infection, and hemorrhage.

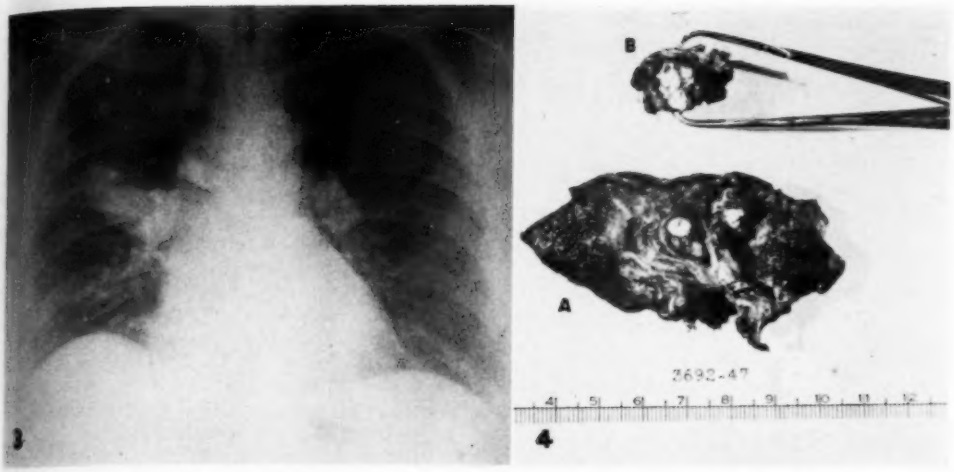
CASE REPORTS

CASE 1: C. B., a 53-year-old white female, was admitted in July 1947, complaining of a sudden pulmonary hemorrhage forty-eight hours before. The hemoptysis continued intermittently until the next evening, when a paroxysmal attack of cough was again accompanied by about one cupful of fresh blood mixed with blood clots. A history was obtained of some pressure pain in the retrosternal region, and some dyspnea of several months duration.

On admission the patient was moderately dyspneic, pale, and perspiring. An emergency portable chest film showed partial consolidation of the right lower lung with atelectasis (Figs. 1 and 2). Multiple calcifications were present in the right hilus and the right paratracheal region. The radiologic diagnostic possibilities considered were bronchial adenoma or carcinoma, broncholith, and bronchiectasis.

The patient continued to bring up 30 to 90 c.c. of fresh blood daily, together with some mucopurulent sputum. No tumor cells could be identified in the expectorated secretions. Bronchoscopy showed only flooding of the bronchi of the middle and lower lobes with blood. In spite of transfusions, the blood pressure dropped from 160/100 to 90/70. The blood count did not go below 4,000,000 red blood cells, the hemoglobin not below 12 gm. The cell volume was 44 per cent. The sedimentation rate was moderately elevated. Skin tests for tuberculosis and coccidioidomycosis were negative.

Since the bleeding could not be stopped by any other means, surgical intervention became necessary. The patient was operated upon by Dr. A. Goldman, who found the right lower and middle lobes to be almost completely atelectatic, with some atelectasis of the base of the upper lobe. Before the thoracotomy, about 1,000 c.c. of blood had to be aspirated by bronchoscopy. A hard calcareous mass,



Figs. 3 and 4. Case 2. The roentgenogram (Fig. 3) shows a dense lesion extending from the right hilus into the lateral portion of the right upper lobe. There is a large calcified paratracheal lymph node on the right, with additional smaller calcifications in the lung lesion.

The surgical specimens (Fig. 4) are (A) the right upper lobe, indurated and atelectatic, containing a bronchiectatic cavity with stones lying loosely in it, and (B) a calcified paratracheal node.

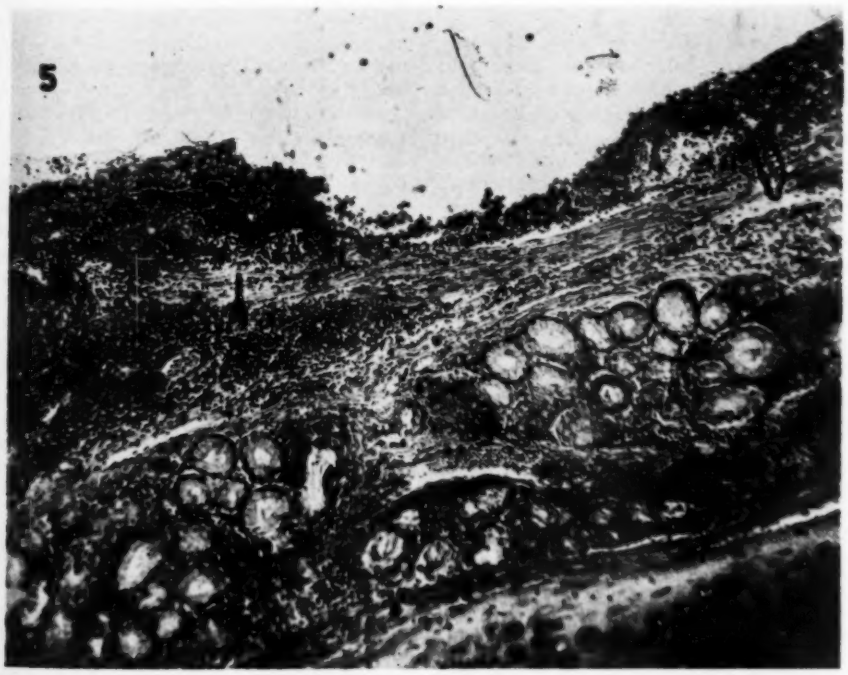


Fig. 5. Case 2. Microscopic section of the ulcerated area in the bronchial mucosa. The floor of the ulcer crater shows infiltration with polymorphonucleated leukocytes, round cells, and much fibrosis.

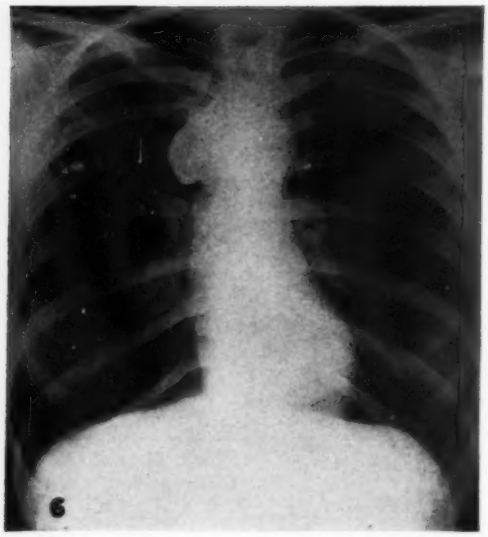


Fig. 6. Case 3. A round radiolucent structure, 2.5 cm. in diameter, is seen in the right subapical region, with a 6-mm. calcification at its floor. There is a calcified paratracheal node on the right.

2 × 3 cm., was found on the medial aspect of the lung, just below the upper lobe bronchus, covered by pleura, which seemed to exert definite pressure on a vessel identified as the anterior basal branch of the right upper lobe artery.

There was softening of the lung around this blood vessel and the calcification, with recent hemorrhage. One area of softening could also be found in the inferior wall of the upper lobe bronchus, where a rock-like exuberance of the calcified mass indented and partially eroded the bronchus. Probing and careful sectioning of the lung specimen, however, failed to reveal any open communication between the bronchus and the blood vessel pressed upon by the calcareous mass, nor could any other possible sources of bleeding be found in microscopic sections. A right pneumonectomy was carried out. The diagnosis, after microscopic study, was focal pulmonary hemorrhage associated with pulmonary calculus.

The patient was discharged in good condition two weeks after the operation.

CASE 2: M. N., a 60-year-old white female admitted in September 1947, gave a history of cough with expectoration of many years duration. About two weeks prior to admission she had noticed bright red blood in the sputum and the hemoptysis had continued. There was no weight loss or chest pain. The past history was not contributory except for "pleurisy" one year before admission, with pain in the right chest aggravated by deep breathing.

Physical examination revealed some decreased breath sounds over the right apex posteriorly but was otherwise negative. Sputum examination and gastric lavage were negative for acid-fast bacilli. Skin tests for coccidioidomycosis and actinomycosis were negative. The sedimentation rate was 33 mm. in one hour; the white blood count was 10,100 with a normal differential count.

A chest film (Fig. 3) and planigrams showed a dense lesion extending from the right hilus into the anterior portion of the right upper lobe, associated with some atelectasis. There was a large calcified paratracheal lymph node on the right. In addition there were several smaller calcifications in the lower portion of the right upper lobe. The findings suggested a chronic inflammatory lesion.

Bronchoscopy by Dr. J. Pressman showed only some thickening and reddening of the bronchial mucosa. A follow-up chest film taken after about two and one-half weeks showed no change in the appearance of the lesion. Clinically a carcinoma of the right upper lobe was suspected.

The patient was operated upon by Dr. A. Goldman, who found a hard, fleshy, atelectatic area in the anterior portion of the right upper lobe. Although it was felt that the mass was probably inflammatory, an upper lobectomy was performed. A mass of paratracheal lymph nodes measuring about 2.0 × 1.0 × 0.75 cm., partially calcified and well pigmented, was removed with the upper lobe. During the peeling off of the upper lobe, one area broke open and about 30 c.c. of yellow pus exuded. An acid-fast stain and a culture of this secretion was negative for tubercle bacilli.

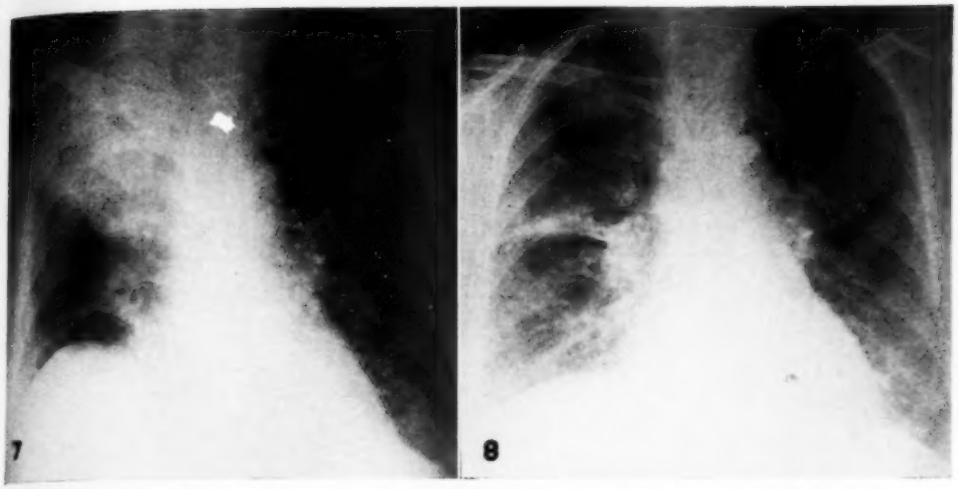
Upon section (Fig. 4), the removed lobe showed much atelectasis, edema, and passive congestion. In the lower portion was a cavity which communicated with the bronchus. The wall of the cavity measured between 0.5 and 1.0 mm. in thickness. Within the cavity were two calcific masses measuring 5 to 7 mm. in diameter. Much regional fibrosis was present.

Microscopic examination showed infiltration with round cells and polymorphonuclears, and areas of bronchial ulceration (Fig. 5). The lining of the cavity consisted of granulation tissue, and the inflammatory changes extended into the surrounding parenchyma. The removed lymph nodes showed anthracosis and calcification. The final diagnosis was focal bronchiectatic abscess of the right upper lobe, containing two broncholiths, with surrounding chronic pneumonitis.

The patient made an uneventful recovery.

CASE 3: F. N., a 33-year-old white female, was admitted for exploratory thoracotomy in July 1947. She gave a history of moderate cough with expectoration, pain in the right upper chest, and hemoptysis for about eleven years. The hemoptysis was usually in the form of blood-tinged sputum, but occasionally massive bleeding occurred. When lying down, the patient often raised 120 c.c. of blood. Running was likely to start a profuse hemorrhage. The chest discomfort varied from mild soreness to sharp pain.

Sputum examination and gastric lavage were neg-



Figs. 7 and 8. Case 4. Fig. 7 shows atelectasis in the right upper lobe with several calcified nodules in the region of the right main bronchus. Fig. 8 is a film taken three weeks after Fig. 7 and one week after expectoration of two broncholiths. It shows almost complete clearing of the atelectasis and a diminished number of calcifications.

ative for tubercle bacilli. Except for slight crackling over the right upper chest posteriorly, the physical examination was negative. A bronchoscopy three weeks prior to admission had failed to reveal the source of the bleeding.

Chest films (Fig. 6) and planigrams showed a round area of diminished density in the right subapical region, measuring about 2.5 cm. in diameter. In the floor of this structure was a calcified body about 14 mm. in its greatest diameter. There was another calcified mass, 3.5 cm. in diameter, in the right paratracheal region. A radiologic diagnosis of a pneumolith associated with either an emphysematous bulla or abscess was made.

On July 25, 1947, the patient was operated upon by Dr. L. Brewer, who excised the lesion in the right upper lobe. The bronchial artery leading to the cystic area measured 4 mm. in diameter. Some adhesions were also freed between the right lung and the calcified mass in the right anterior mediastinum.

Gross examination of the specimen showed a 2-cm. abscess cavity containing an irregular calculus measuring 12 mm. The average thickness of the abscess wall was 4 mm. and the inner surface of the cavity was ulcerated and hemorrhagic. It was covered by a layer of amorphous cellular debris and leukocytes. Beneath this layer was granulation tissue heavily infiltrated with small round cells, plasma cells, multinucleated giant cells, and large mononucleated phagocytes. The adjacent alveoli and bronchioles showed some fibrous scarring of their walls, some round cell infiltration, and epithelial metaplasia. There was no evidence of inflammatory changes of a specific histologic character. The diagnosis, made by Dr. A. Wright, was chronic lung abscess with pulmonary calculus.

The patient was discharged in good condition eight days after the operation and has been entirely free from signs and symptoms since.

CASE 4: C. B., a 65-year-old white female, was admitted in July 1947, complaining of fatigability, non-productive cough, dyspnea on slight exertion, and anorexia for about one month. Two days prior to admission, she had an attack of chest pain in the substernal region, with pallor and mild cyanosis.

Physical examination showed no cardiac murmurs, but occasional skipped beats and possible slight cardiac enlargement. Moist râles were heard at both bases, particularly on the right side. There was definite electrocardiographic evidence of a recent posterior coronary occlusion.

A portable chest film (Fig. 7) showed mild pulmonary hyperemia and some emphysema. The greater portion of the right upper lobe was atelectatic, with displacement of the trachea to the right. There were several irregular areas of calcification, measuring 4 to 8 mm., in the medial portion of the right upper lobe, near the hilus, which were not considered to be of significance. The radiologic diagnosis was atelectasis of the right upper lobe, probably secondary to a bronchial carcinoma.

Two further chest x-ray examinations, about a week apart, failed to reveal any evidence of clearing in the right upper lobe. After a total stay of three weeks in the hospital, the patient was discharged, the plan being to reinvestigate the pulmonary condition as soon as she had recovered from the heart attack.

A few days after her discharge, the patient coughed up an irregular, hard, rock-like concretion, measuring 2 × 3 × 4 mm., which after decalcification revealed a nucleus containing organic debris

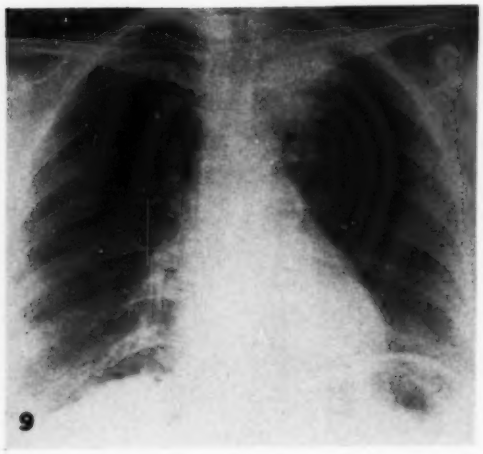


Fig. 9. Case 5. Inverted pear-shaped density in the left upper lobe, with numerous radiolucent areas. There are several small calcifications near the lower border of the lesion.

Fig. 10. Case 5. Planigram demonstrating a close relationship of the calcifications to the upper lobe bronchus and radiolucent areas within the consolidation.

with anthracotic pigmentation. The pathologist's description was "a broncholith, probably the portion of a calcified peribronchial lymph node."

A chest film (Fig. 8) taken after expulsion of the stone showed almost complete clearing of the right upper lobe, with re-expansion and with disappearance of a small calcification seen previously at the apex of the atelectatic shadow.

Upon further questioning, the patient stated that, while still in the hospital, she had coughed up and subsequently swallowed what she thought was another small "pebble."

CASE 5: E. E., a 53-year-old white female, was admitted to the hospital in October 1947. Her chief complaints were cough, occasional hemoptysis, and shortness of breath, the onset of which dated back to an acute pulmonary illness about five months earlier, diagnosed as pneumonia. During the week prior to admission her temperature ranged up to 102°, with frequent chills and with expectoration of half a cup of mucosanguineous fluid daily, frequently mixed with blood clots.

The respiratory rate was 30; breathing was labored, independent of position. Physical examination of the chest showed slight wheezing over the left upper lobe. The leukocyte count was 17,000 and there was a mild secondary anemia.

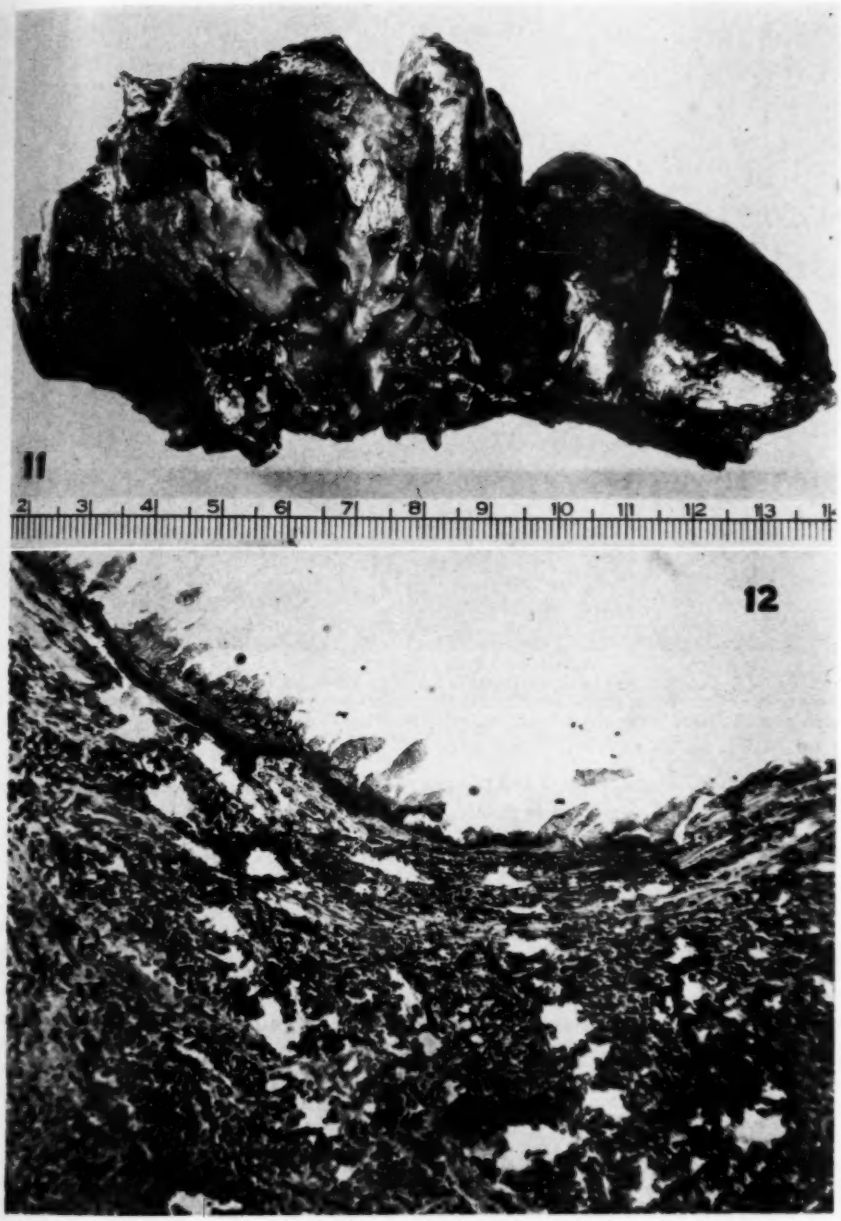
Chest films and planigrams (Figs. 9 and 10) revealed a sharply defined, inverted, pear-shaped density, 6.0 × 3.5 cm., in the apical portion of the left upper lobe, within which were seen a few small, irregular, radiolucent areas. Near the lower border of the lesion, and in close relationship to a large bronchus leading into the diseased portion of the left upper lobe, a few small calcifications were present. Laminagrams showed these calcifications to be situated in the bronchial lumen. The suggested x-ray diagnosis was pulmonary suppuration, probably secondary to broncholiths.

Bronchoscopy showed purulent drainage from the left upper lobe bronchus, which on examination was negative for tumor cells or tuberculosis.

The patient was operated upon by Dr. A. Goldman, who found the apical segment of the left upper lobe to be firm and atelectatic, with numerous adhesions to the first two ribs. A segmental resection was performed.

Macroscopic examination (Fig. 11) of the specimen showed an area of calcification firmly adherent to the wall of the upper lobe bronchus and protruding into its lumen. The bronchus was considerably narrowed at this point, and distal to it bronchiectatic changes were present. Several calcified lymph nodes, up to 1 cm. in diameter, were found close to the proximal portion of the bronchus. Lying loose in the posterolateral branch of the bronchus there were two irregular broncholiths, measuring 3 × 3 × 5 mm.

The microscopic sections showed chronic inflammation, round-cell infiltration, much anthracosis and fibrosis, bronchiectatic dilatations, and parenchymal atelectasis. Within the bronchioles and alveoli a mucopurulent exudate was present, containing xanthoma cells and erythrocytes. There was evidence of some columnar and osteoid metaplasia, with cal-

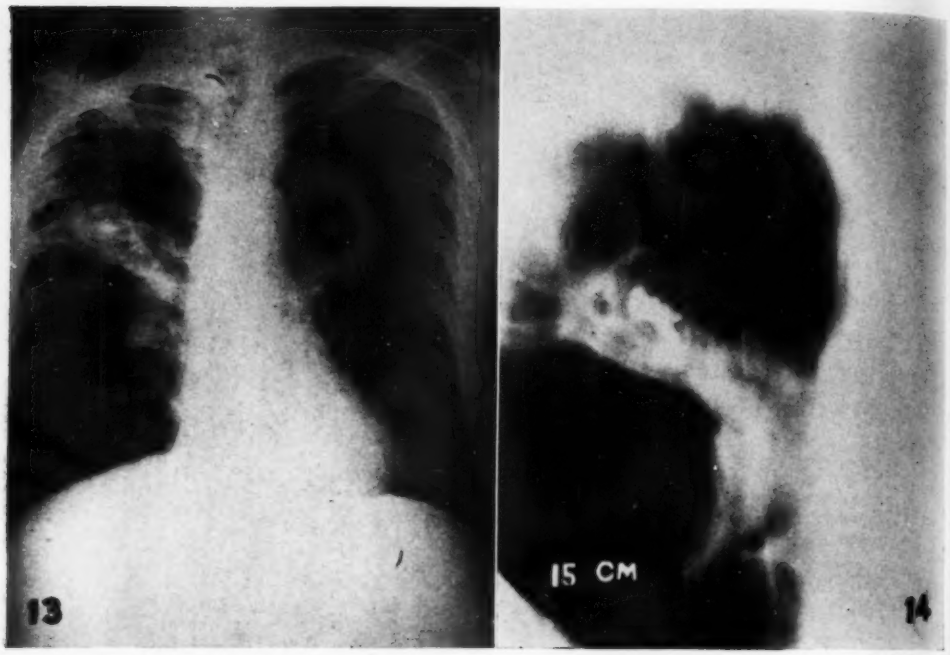


Figs. 11 and 12. Case 5. Opened specimen of the right upper lobe (Fig. 11) shows multiple bronchiectatic cavities, from which two stones were removed. The photomicrograph (Fig. 12) shows an abscess cavity, the lining of which contains many calcium particles. Under these (left side of photograph) some osteoid tissue can be seen with osteoblasts, indicating bony metaplasia.

cium particles lining some of the ulcerated areas (Fig. 12). There was no evidence of neoplasm or of active tuberculosis.

Postoperative empyema developed and the patient died two weeks after the operation, of a massive pulmonary hemorrhage.

CASE 6: W. S., a 57-year-old white male, was admitted in October 1948 with a history of repeated



Figs. 13 and 14. Case 6. Fig. 13 shows an area of density extending from the right hilus to the lateral portion of the right upper lobe. There are calcifications in the central part of the lesion. The planigram (Fig. 14) shows multiple radiolucent areas in the central part of the lesion. It also shows the calcification to be a single staghorn structure.

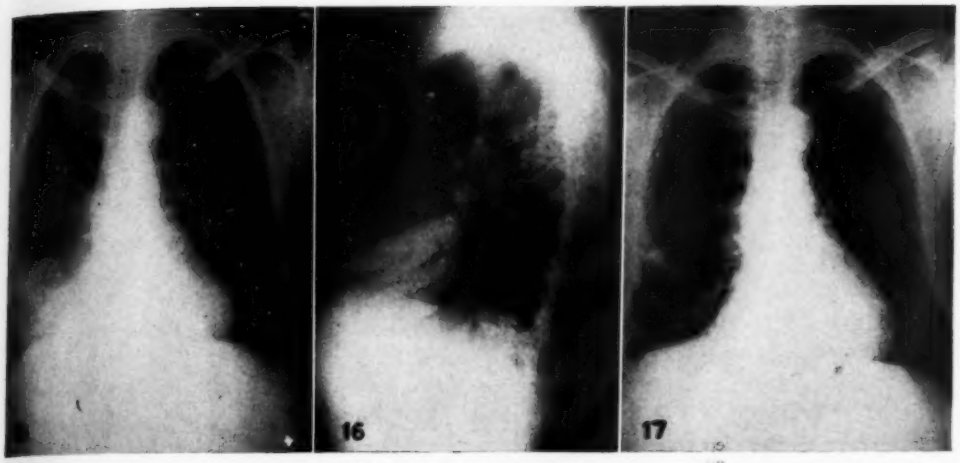
pebrile attacks, hemoptysis and acute pneumonitis since 1922, at which time he was thought to have an acute lung infection, possibly of influenzal nature. He had suffered from chronic productive cough ever since. The sputum was thick and greenish, with an offensive odor, and was negative for tubercle bacilli. The acute illnesses always responded well to antibiotics and chemotherapy. During a recent flare-up the white count had been 17,100, with 70 per cent polymorphonuclears; the temperature had subsided after three days. Bronchoscopy at that time showed moderate edema and redness of the right upper lobe orifice, from which some mucopurulent material was seen to drain. No evidence of neoplasm was found. Papanicolaou stain was negative for tumor cells.

Chest films and planigrams (Figs. 13 and 14) showed a densely consolidated area in the basal portion of the right upper lobe with central calcifications and small areas of ulceration.

The patient was operated on by Dr. A. Goldman, who found a chronic pulmonary abscess in the right upper lobe with small interlobar accumulations of thick, granular pus. A lobectomy was carried out extrapleurally, because of the dense adhesions surrounding the right upper lobe. On cutting the specimen, a staghorn-shaped broncholith about 1 cm. in greatest length was found within a large bronchus just proximal to the abscess cavity. Surrounding this bronchiectatic abscess were chronic pneumonitis and atelectasis. The microscopic sections showed ulceration of the bronchial mucosa, thickening of the bronchial wall, and hyperplasia of the mucous glands. The final pathological diagnosis was broncholith with bronchiectatic abscess, chronic pneumonitis, and pleural adhesions.

CASE 7: On admission in July 1948, A. D., a 58-year-old white male, gave a history of cough with production of a moderate amount of sputum ever since a "virus infection" in January 1948. He had lost 18 pounds of weight since that time and during the last four weeks had chills, night sweats, and fever.

The admission temperature was 102.8°. The white blood count was 11,200 with 87 per cent polymorphonuclears. The patient complained of some pain in the right lower chest aggravated by coughing, and râles were heard anteriorly and posteriorly at the right base, with some dullness to percussion. The x-ray examination (Figs. 15 and 16) showed atelectasis of the right middle lobe with some involvement of the right lower lobe. There were several calcifications in the right hilar region. Although neoplasm could not be ruled out with certainty, the diagnosis of a chronic inflammatory lesion due to broncholithiasis was favored.



Figs. 15–17. Case 7. Fig. 15 shows atelectasis of right middle lobe, with small areas of calcification in right hilus. In Fig. 16, a lateral view, some of the areas of calcification are seen to be close to the right middle lobe bronchus, Fig. 17, a film taken three weeks after bronchoscopic removal of broncholiths from the right main bronchus, shows almost complete clearing of atelectasis.

Bronchoscopy showed edema and narrowing of the right main stem bronchus up to and beyond the middle lobe bronchus. Some bleeding was noted from the stenotic middle lobe orifice, and a biopsy was done. It was reported as showing chronic bronchitis with some squamous metaplasia. Papanicolaou stain was negative for tumor cells. There were some pus cells and mucus, without evidence of acid-fast bacilli or fungi on cultures.

At thoracotomy, the right lung showed many adhesions, with pockets of purulent pleural fluid. A pneumolysis was performed. Dissection of the hilus showed no tumor, but revealed a large calcified lymph node pressing on the right lower lobe bronchus, with partial atelectasis of the middle and lower lobes. The thoracotomy procedure was interrupted at this point and a bronchoscopy was performed. Several calcified broncholiths were seen in the lumen of the right lower bronchus and were removed.

A follow-up bronchoscopy after two weeks showed a few fresh granulations at the point of the previous perforations, with slight narrowing of the bronchial lumen. The granulations were broken up by sponge, and the bronchus was dilated.

The postoperative clinical course was uneventful, and the follow-up chest films (Fig. 17) showed progressive clearing. The final diagnosis was chronic pneumonitis with partial atelectasis of the right middle and lower lobes, and empyema, secondary to bronchial erosion by calcified hilar lymph nodes.

CONCLUSIONS

1. Seven cases of active bronchopulmonary lithiasis are reported, of which 6 were proved at surgery and subsequent pathologic examination, while in the seventh the stones were expectorated. This raises the total number of cases reported in the American and English literature (since 1900) to 103.

2. Active bronchopulmonary lithiasis is supposedly a rare clinical syndrome, but probably occurs more often than is suspected clinically. It should be included in the differential diagnosis of every case of bronchial obstruction, pulmonary suppuration, and hemorrhage associated with paroxysmal attacks of cough, where the chest roentgenogram shows calcific shadows in the region of consolidated areas.

3. Broncholiths may develop within the bronchi or may originate outside the bronchi, with subsequent perforation into the air passages. In the majority of cases broncholiths are due to perforation of calcified tuberculous lymph nodes.

4. The x-ray changes found in broncholiths may simulate carcinoma of the lung, chronic lung abscess, bronchiectasis with atelectasis, chronic pneumonitis, as well as fungoid disease.

5. Laminagraphy is an important diagnostic aid in bronchopulmonary lithiasis, as it may demonstrate the stone perforating the bronchial wall or lying in the lumen of the bronchus.

6. Early recognition and treatment of

this condition may prevent the serious complications caused by bronchial obstruction, pulmonary suppuration, and hemorrhage.

Cedars of Lebanon Hospital
Los Angeles 27, Calif.

We are greatly indebted to Drs. A. Goldman, S. Strouse, C. Strouse, M. Fink, and L. Brewer for their permission to use the clinical records of their cases for this paper, and also to Dr. H. Goldblatt for discussing some of the microscopic sections with us.

REFERENCES

1. ANDERSON, W. S., AND MACKAY, J. B.: Broncholithiasis. Dis. of Chest **10:** 427–432, 1944.
2. ATLEE, L. W.: Bronchial Concretions. Am. J. M. Sc. **122:** 49–54, 1901.
3. AUERBACH, O.: Perforation of Tuberculous Lymph Nodes into the Trachea and Bronchi. Arch. Otolaryng. **39:** 527–532, 1944.
4. BARR, D. P., AND BULGER, H. A.: Clinical Syndrome of Hyperparathyroidism. Am. J. M. Sc. **179:** 449–476, 1930.
5. BARRETT, J. H.: Broncholithiasis. Arch. Otolaryng. **44:** 574–580, 1946.
6. BLADES, B., AND GRAHAM, E. A.: Surgical Treatment of Intractable Pulmonary Hemorrhage. New Internat. Clin. **4:** 77–83, 1939.
7. BROWN, L. E.: Pneumoliths. Report of Two Cases. Cleveland M. J. **16:** 21–25, 1917.
8. CHANDLER, F. G.: Lung Stone Causing Profuse and Recurrent Haemoptysis. Lancet **2:** 13–14, 1943.
9. CHAPPELL, W. F.: History of a Broncholith, Bronchial Calculus, or Lung Stone. M. Rec. **84:** 294, 1913.
10. CRANE, A. W.: Pulmonary Metastasis. Am. J. Roentgenol. **5:** 479–482, 1918.
11. DAWSON, J. W., AND STRUTHERS, J. W.: Generalised Osteitis Fibrosa, with Parathyroid Tumour and Metastatic Calcification. Edinburgh M. J. **30:** 421–564, 1923.
12. ELLIOTT, A. R.: Broncholithiasis. J. A. M. A. **79:** 1311–1314, 1922.
13. FARR, C. B.: Bronchial Calculi, with Report of a Case. Internat. Clin. **4:** 288–294, 1908.
14. FOX, H.: Pneumolith or Broncholith. J. A. M. A. **80:** 175–176, 1923.
15. FOX, J. R., AND CLERF, L. H.: Broncholithiasis. Ten Cases. Ann. Int. Med. **23:** 955–959, 1945.
16. GANDER, G.: Ein Beitrag zur Frage der verästelten Lungenverknöcherungen. Fortschr. a. d. Geb. d. Röntgenstrahlen **44:** 448–451, 1931.
17. GOLDBLATT, H.: Personal communication.
18. GRAHAM, E. A., SINGER, J. J., AND BALLON, H. C.: Surgical Diseases of the Chest. Philadelphia, Lea & Febiger, 1935, pp. 564–567.
19. HARBITZ, F.: Extensive Calcification of the Lungs as a Distinct Disease. Arch. Int. Med. **21:** 139–146, 1918.
20. JACKSON, C.: Bronchoscopy in Broncholithiasis. Tr. Am. Acad. Ophth. & Otol., 1923, pp. 352, 354.
21. JACKSON, C., AND JACKSON, C. L.: Bronchoscopy, Esophagoscopy, and Gastroscopy. Philadelphia, W. B. Saunders Co., 1934, p. 323.
22. JACKSON, C., AND SPENCER, W. H.: A Broncholith and Pneumoconiotic Material Removed from a Bronchiectatic Cavity by Peroral Bronchoscopy. New York M. J. **113:** 461–462, 1921.
23. KYLE, D. B.: Broncholithiasis, with Report of Case. Laryngoscope **14:** 711–713, 1904.
24. LEROY, L.: Quoted by Atlee (2). Les concrétions bronchiques. Thèse pour le doctorat en Médicine, Paris, 1868.
25. LLOYD, J. J.: Broncholiths, with Report of 4 Cases. Am. J. M. Sc. **179:** 694–699, 1930.
26. LYTER, J. C.: Bronchopulmonary Lithiasis. M. Clin. North America **6:** 107–117, 1922.
27. Massachusetts General Hospital Case 32302. New England J. Med. **235:** 139–142, 1946.
28. MAYTUM, C. K., AND VINSON, P. P.: Pulmonary Calculus Simulating Primary Bronchial Carcinoma. Report of Case. M. Clin. North America **15:** 1551–1553, 1932.
29. MYERS, D. W.: Broncholithiasis. Dis. of Chest **6:** 269–272, 1940.
30. NAGER, F. R.: Ueber Broncholithiasis. Schweiz. med. Wchnschr. **73:** 6–8, 1943.
31. PENDERGRASS, E. P., AND DE LORIMIER, A. A.: Broncholiths and Stone Asthma. Radiology **25:** 717–722, 1935.
32. POULALION, S. A. M.: Quoted by Lloyd (25). Les pierres du poumon, de la plèvre et des bronches, et la pseudo-phtisie pulmonaire d'origine calculeuse. Paris, G. Steinheil, 1891, p. 364.
33. PRATTEN, F. H.: Lung Stones (Pneumoliths) Possibly Associated with Tuberculosis. Canad. M. A. J. **17:** 216–217, 1927.
34. PRITCHARD, J. S.: Some Interesting Cases of Calcareous Degeneration Found in the Thorax. Arch. Int. Med. **32:** 259–282, 1923.
35. BOBB, J. B.: Bronchial Calculus. Report of Case. Iowa M. J. **14:** 474, 1908.
36. RUBIN, E. H.: Diseases of the Chest, with Emphasis on X-Ray Diagnosis. Philadelphia, W. B. Saunders Co., 1947, pp. 372–373.
37. SALINGER, H.: Multiple Knochenbildung in der Lunge. Fortschr. a. d. Geb. d. Röntgenstrahlen **44:** 793, 1931.
38. SALINGER, H.: Die Knochenbildungen in der Lunge mit besonderer Berücksichtingung der Tuberösen Form. Fortschr. a. d. Geb. d. Röntgenstrahlen **46:** 269–275, 1932.
39. SCHENCK, J.: Quoted by Barrett (5). Observationum medicarum. Frankfurt, sumpt. J. Rhodii, 1600, Vol. 1, p. 351.
40. SCHMIDT, H. W.: Broncholithiasis. Proc. Staff Meet., Mayo Clin. **13:** 609–612, 1938.
41. SHASTID, W. E.: Bronchitis of Unusual Type. Illinois M. J. **47:** 67–69, 1925.
42. STIVELMAN, B. P.: Broncholithiasis. Am. Rev. Tuberc. **18:** 430–434, 1928.
43. TINNEY, W. S., AND MOERSCH, H. J.: Broncholithiasis. S. Clin. North America **24:** 830–838, 1944.
44. VAN ORDSTRAND, H. S., MOORE, M., JR., AND HARRIS, H. E.: Broncholithiasis. Report of 2 Cases. Cleveland Clin. Quart. **9:** 36–44, 1942.
45. VINSON, P. P., AND BUMPUS, L. D.: Pneumoliths Producing Pulmonary Suppuration. Seven Cases. M. Clin. North America **15:** 79–85, 1931.
46. VINSON, P. P., AND PEMBLETON, W. E.: Ulceration of Tuberculous Lymph Node into Lumen of Bronchus, with Bronchoscopic Removal. Report of Case. Ann. Otol., Rhin. & Laryng. **49:** 797–800, 1940.
47. WELLS, H. G.: Metastatic Calcification. Arch. Int. Med. **15:** 574–580, 1915.
48. WELLS, H. G.: Calcification and Ossification. Arch. Int. Med. **7:** 721–753, 1911.
49. ZAHN, D. W.: Broncholithiasis. Am. Rev. Tuberc. **54:** 418–423, 1946.
50. ZWEIFEL, E.: Über zwei Fälle von Broncholithiasis. Zur Differentialdiagnose der Bronchospirochaetosis Castellani. Schweiz. med. Wchnschr. **74:** 833–835, 1944.

SUMARIO

Litiasis Broncopulmonar Activa

De los 7 casos de litiasis broncopulmonar activa comunicados, 6 fueron comprobados al operar y en el subsiguiente examen patológico, y en el otro se expectoraron los cálculos. Con éstos, se eleva a 103 el total de casos comunicados (desde 1900) en la literatura estadounidense e inglesa.

La litiasis broncopulmonar activa pasa por ser un síndrome clínico raro, pero con toda probabilidad es más frecuente que lo que se sospecha clínicamente, debiendo figurar en el diagnóstico diferencial de todo caso de oclusión bronquial, supuración pulmonar y hemorragia asociada a ataques paroxísticos de tos, siempre que la radiografía torácica revele sombras de calcificación en las zonas de hepatización.

Los broncolitos pueden formarse dentro de los bronquios o tener su origen fuera de ellos, con perforación después a las vías aéreas, debiéndose, en la mayoría de los casos, a perforación por ganglios linfáticos tuberculosos calcificados.

Las alteraciones reveladas por los rayos X en los broncolitos pueden simular carcinoma pulmonar, absceso pulmonar crónico, bronquiectasia con atelectasia, neumonitis crónica y hasta micosis.

La laminografía constituye un importante auxiliar diagnóstico en la litiasis broncopulmonar, pues puede revelar el cálculo perforando la pared bronquial o descansando en la luz del bronquio.

El reconocimiento y tratamiento tempranos del estado pueden impedir las graves complicaciones ocasionadas por la obstrucción bronquial, la supuración pulmonar y la hemorragia.

DISCUSSION

William M. M. Kirby, M.D. (San Francisco, Calif.): This has been a comprehensive and clear-cut presentation of a subject of importance to all concerned with the diagnosis and treatment of pulmonary diseases. It can certainly be agreed that bronchopulmonary lithiasis is more common than the 100 odd cases in the literature would indicate. This is particularly true of the more benign varieties; in any large chest center a few patients can be found with a collection of stones they have expectorated.

The cases presented illustrate clearly the difficulties often encountered in diagnosis and management, and this paper will serve to remind us of bronchopulmonary lithiasis in our encounters with obscure pulmonary problems.

"Egg Shell" Calcifications In Silicosis[1]

CHARLES E. GRAYSON, M.D., and HELEN BLUMENFELD, M.D.

CHARACTERISTIC calcium densities of apparently unique morphology occur in chest roentgenograms of men having silicosis. These shadows are seen as more or less regular rings in the hilar or mediastinal regions of the chest. They have been designated as egg-shell, *Eierschalen*, or mulberry calcifications. They are circular or ovoid in character and consist of an irregular peripheral shell with a faint stippling throughout the enclosed tissue.

Considerable divergence of opinion exists as to the cause and significance of this particular formation. There has, unfortunately, been a paucity of autopsy studies to clarify the problem.

Lommel (2) was of the opinion that calcium is deposited in inflamed and dilated sinuses within the lymph node capsule and other similarly dilated lymphatic spaces. He states that similar calcifications are seen in the extreme periphery of the lung. He also mentions fine, delicate ring forms which may be early or transitional stages of the same process. Mild cases with "snow-storm" lung changes sometimes show the calcifications very clearly. Lommel reports a 20 per cent incidence of calcifications in cases of silicosis without clinically proved tuberculosis.

Sweany *et al.* (10) show that the annular densities are calcareous and express the opinion that, "in cases of silicosis, such a formation represents collateral infection, chiefly by the tubercle bacillus." Sweany (9) does state that the lymph node changes are characteristic and identifies the subcapsular location of the calcium infiltration.

Schulte and Husten (7) were of the opinion that the shadows represent dense silicotic connective tissue around bronchi, extending into the lung fields.

Davies (1) presents twenty-three cases and discusses the probabilities. (*a*) Calcification exists, in which case it is due to silico-tuberculosis and must be associated with tuberculous infection. It may be due to a degenerative process following a silicotic infiltration of the lung root glands attracting lime salts, which become deposited in them. (*b*) Calcification is not present, the shadows representing silicotic nodules in the lung itself.

Riemer (4) is of the opinion that the densities may represent the results of inhaled calcium along with the silica dust. He presents four cases having no clinical evidence of tuberculosis. In one the tuberculin reaction was negative.

Rigler (5) refers to the calcification of the lymph nodes in silicosis as "classic."

The present study is a correlation of history, clinical and roentgenographic findings, and histopathology, in an attempt to produce a clearer picture of this phase of silicosis and to determine the significance of the unique morphology of the calcium deposits.

PRELIMINARY REMARKS

Preliminary remarks will aid in a more critical evaluation of the material and data, which were obtained at the San Francisco City and County Hospital. Many cases were seen in the Tuberculosis Division and the Chest Clinic, as well as in the medical and surgical wards. This variation accounts for a large number of tuberculous silicotics but also supplied many patients incidentally admitted with totally unrelated and frequently fatal diseases. The preponderance of older men furnished the milder cases of long duration mentioned by Lommel. The gamut of conditions for which these patients were

[1] From Stanford University School of Medicine, Departments of Radiology and Pathology, San Francisco Hospital, San Francisco, Calif. Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

TABLE I: STATISTICAL CORRELATION OF DATA IN 88 CASES OF SILICOSIS

	Number of Cases	Average Age (years)	Average Exposure Time (years)	Average Time Since Earliest Exposure (years)	Number of Cases with Autopsy	Tuberculosis		
						Active	Arrested	No Evidence
Total	88	57	19	33	39	38	3	47
Egg-shell calcifications	37	58	16	34	17	15	0	22
No egg-shell calcifications	51	57	21	32	22	23	3	25

seen included respiratory infections (acute pneumococcal pneumonias, coccidioidomycosis, tuberculosis), heart disease (arteriosclerotic heart disease, cor pulmonale, etc.), neoplasms (carcinomata of the alimentary and respiratory tracts), and a few of the less frequent diseases.

The derivation of our cases from mining territory with less crowded population may be of significance in the low incidence of tuberculous silicotics. Thus the distribution and character of the silicotic changes as a whole in our group should not be considered representative. On the contrary, these were generally milder processes which had incapacitated the individual only moderately or not at all and had been usually incidental to some other condition.

The diagnosis of silicosis was made by various radiologists, internists, and pathologists on the basis of roentgenograms, history and, when possible, histology. Many cases which were seen could not be included because the history was inadequate or roentgenograms were unavailable for review, despite the fact that roentgenographic and histologic reports were adequate. The peculiar calcifications, though present, were recorded by the roentgenologist in very few instances.

Obviously, it is impossible to determine the severity of exposure without knowing its approximate intensity and duration, the particle size and other critical factors. Nevertheless, some information may be obtained from statistical study of data.

DATA

Two hundred cases of silicosis were reviewed. In 114 of these, chest roentgenograms and recorded data were available. The 60 autopsies of the latter group confirmed the diagnosis of silicosis in every instance. In 88 cases there were sufficient data for approximate dust exposure statistics (Table I).

There was a total of 40 cases with egg-shell calcifications. In 3 instances there was only a note that the man had been a hard rock miner and these cases were not included in the group of 88. None of these 3 showed any clinical evidence of tuberculosis. In 1 there was histologic evidence of arrested tuberculosis and 1 had no histologic evidence of tuberculosis at postmorten examination. Autopsy was not done on the third.

The 37 cases with egg-shell calcifications are compared with the remaining 51 having no visible hilar calcifications. It is to be noted that the average age is essentially the same in both groups. The group with calcifications showed an average of five years less total exposure to silica and a two-year longer interval since the beginning of exposure. These findings support the belief that the milder (not minimal) instances of silicosis are to be seen in those who live longer and therefore deposit calcium many years after the original exposure.

Sixty per cent of those having calcifications and 49 per cent of those having no calcifications were without evidence of tuberculosis. Incidentally, the 3 cases of arrested tuberculosis were without calcification, and the arrested tuberculosis was found postmortem. Thus there was an 11 per cent greater incidence of tuberculosis in those cases without calcification.

The important group, though small, is

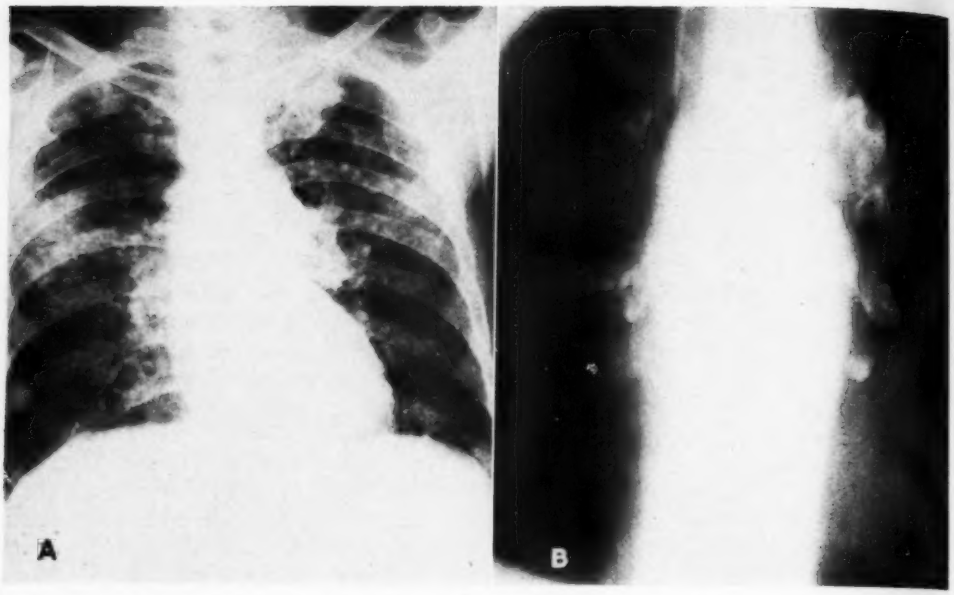


Fig. 1. Male, age 57, an "axe grinder" for two years beginning twenty years before. No exposure to silica dust for past eighteen years. Genito-urinary tuberculosis in hospital.
A. Note scattered pulmonary nodules containing punctate calcium deposits.
B. Tomogram reveals more nodes and demonstrates shell characteristics. Main bronchi visible.

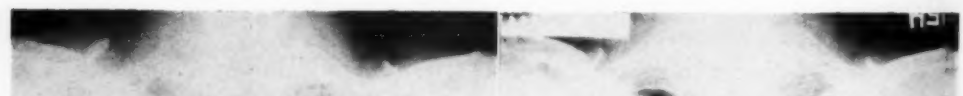


Fig. 2. Male, age 50, hard rock miner for seventeen years. No exposure for thirteen years. No clinical evidence of tuberculosis.
A. Note enlarged hilar nodes without visible calcifications.
B. Calcifications present four years later. Still no clinical evidence of tuberculosis.

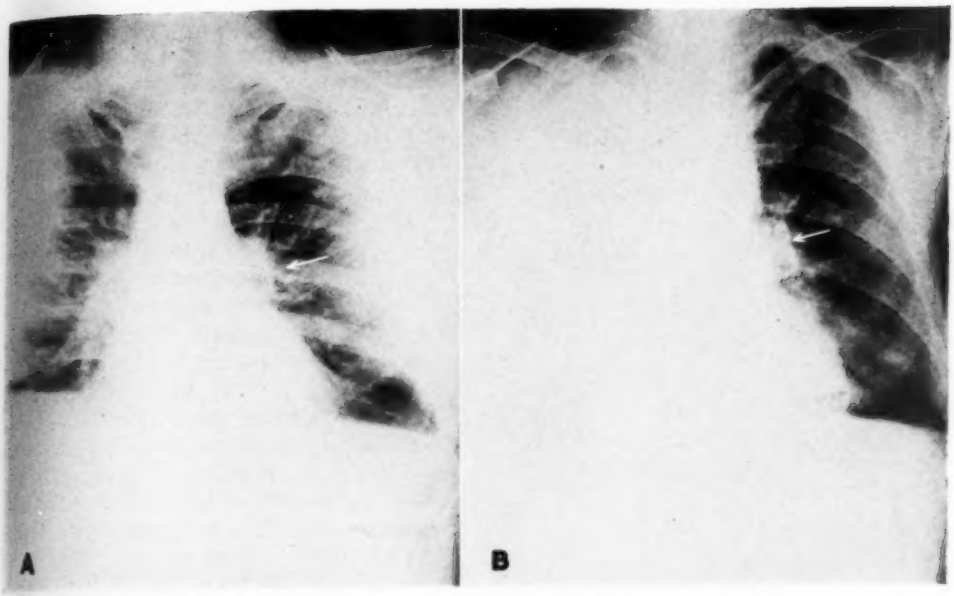


Fig. 3. Male, age 52, hard rock miner for six years. No exposure for twenty-four years.
A. Pulmonary silicotic changes. Thick pleura with pleural calcium plaque on right.
B. Ten years later egg-shell calcifications are demonstrable in the left hilus. Entire right chest opaque. Autopsy revealed no evidence of tuberculosis. Primary pleural neoplasm in right chest.

that of 8 cases in which there were calcifications of characteristic morphology but no evidence of tuberculous or any other chronic pulmonary infection clinically or histologically. The criteria for selection of these 8 cases should be reiterated. There was a history of exposure to silica. The roentgenograms showed shell calcifications and nodular pulmonary densities. The diagnosis of silicosis was confirmed at autopsy. There was no clinical or histological evidence of chronic pulmonary infection. Further proof of the relationship between silicosis and "egg-shell" calcifications seems unnecessary.

Several individual cases lend emphasis to the tabular data and illustrate, in detail, the stages in development of the calcific process. Three men having heavy calcifications were "grinders" with about two years exposure twenty, twenty-two and thirty-four years before examination (Fig. 1). This emphasizes the time factor following exposure rather than its duration. This is in accord with Lommel's

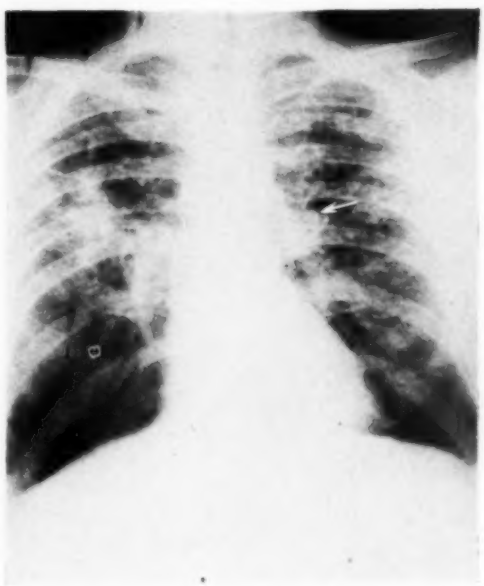


Fig. 4. Male, age 48, quartz miner for over one year twenty-two years before; Blacksmith twenty years. No clinical evidence of tuberculosis. Nodular pulmonary densities becoming confluent in right midlung field. Small shell calcifications in hilar regions.

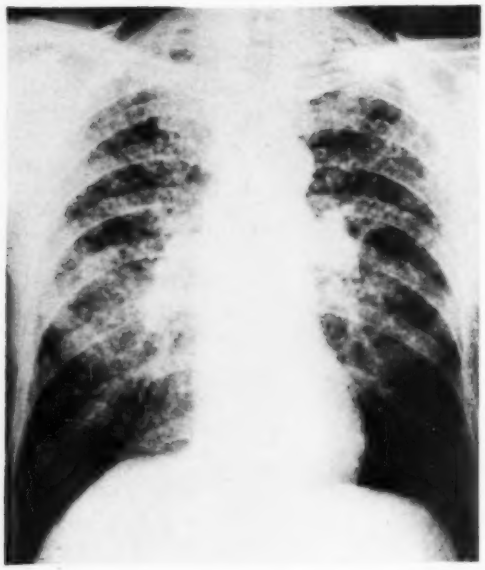


Fig. 5. Male, age 62, hard rock miner for thirteen years. No exposure for next fifteen years. Peripheral calcifications, all granular and not shell-like. No clinical or histologic evidence of tuberculosis.

remarks. It is noteworthy that the amount of calcium from a grinding wheel is certainly minimal. This would weaken Riemer's suggestion that the calcium might be inhaled. Furthermore, inhaled calcium has been reported to disappear from the lungs in a few months (3).

The development of calcification in nodes many years after exposure is well demonstrated in a case in which uncalcified nodes were previously visible in chest roentgenograms (Figs. 2 and 3). Calcification does occur in confluent third-stage silicosis (Fig. 4), though infrequently. The "snow-storm" lungs of silicosis with definite peripheral calcification are seen less frequently (Fig. 5). The parenchymal calcifications are *not* shell-like, as Lommel indicates. Shell calcifications are found in distant sites (Fig. 6) only where there were pre-existing lymph nodes (6), but not in the lung parenchyma. These may occur moderately far out in the lung fields but in close relation to the bronchi (Fig. 7).

The correlation of the clinical and post-

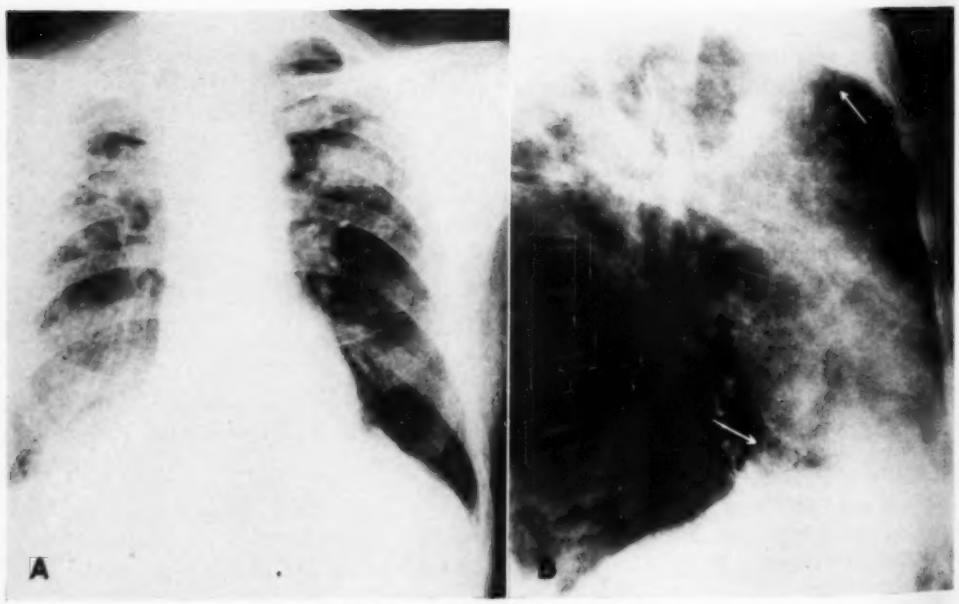


Fig. 6. Male, age 67, hard rock miner for five years. No exposure for past twenty-six years.
A. Note pulmonary densities without visible perihilar calcified nodes.
B. Lateral projection showing the characteristic calcification behind the manubrium and in the posterior cardiophrenic angle.

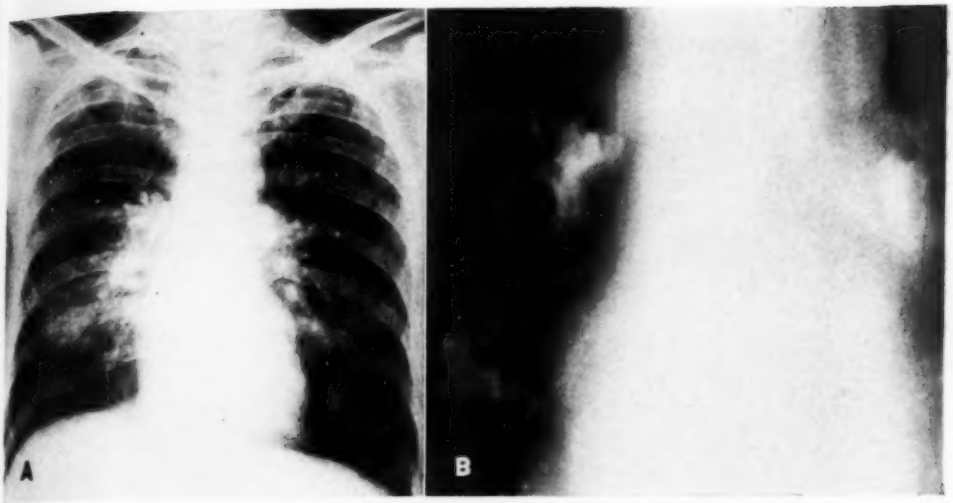


Fig. 7. Male, age 60, hard rock miner twenty-five years. No exposure in past ten years. No clinical evidence of tuberculosis.
A. Mild pulmonary nodulation. Heavy hilar shell calcifications. Note pleural calcium plaques near apices.
B. Tomogram showing peribronchial distribution and shell characteristics.

mortem studies in those few silicotics without demonstrable tuberculosis is best represented by one case (Fig. 8). Like most of the other silicotics, this man had been a "hard-rock miner" for twenty years, beginning forty-one years before; he had been a farmer for the following nineteen years, and for the past two years a night watchman. He was "a little more short of breath than he used to be" and he had some recent "chest colds." He was cachectic when he entered the hospital, with complete bowel obstruction. His fingers were moderately clubbed. He died three days after admission. Postmortem examination showed adenocarcinoma of the cecum with abdominal carcinomatosis. Selected parts of the autopsy report are quoted:

"The aortic lymph nodes are enlarged, hard and pigmented a bluish-grey. The (mediastinal) lymph nodes are enlarged . . . the hilar lymph nodes are greatly enlarged, very firm and blue-black. On cut section they show mottled white areas in a blue-black matrix and there are often seen concentrically arranged layers of white tissue within the substance of the node (Fig. 9A). The right ventricle is not enlarged. The right lung weighs 1,385 gm. and the left 1,020 gm. Both lungs are dark reddish-purple and

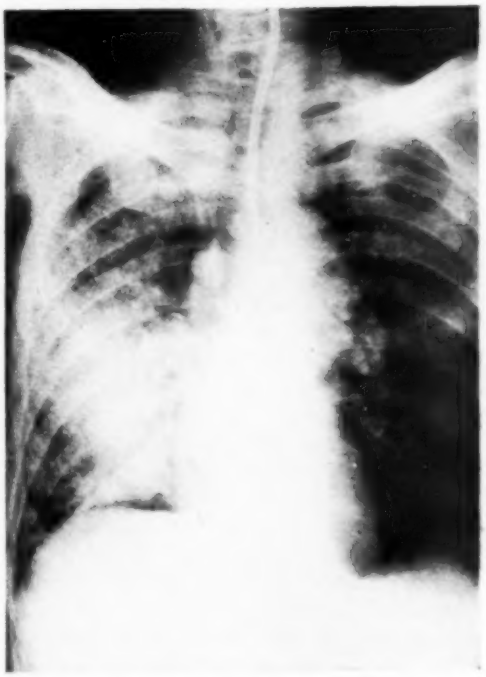


Fig 8. Male, age 63, hard rock miner for twenty years. No exposure in past twenty-one years. Acute bowel obstruction. Acute pneumonia in right lung (verified postmortem). Note heavy hilar shell calcifications.

show black mottled areas on their surfaces. On both lungs are severeal puckered areas which are the result of scarring. Within the lungs are numerous small irregularly shaped areas which are extremely hard and feel like small bits of gravel. These are quite uniformly distributed throughout both lungs and are most marked in the apices, where some areas seem to consist of confluent material of the above sort. On cut section . . . these . . . hard areas are seen to be grey, varying from 0.1 to 3 or 4 mm. in size. The gallbladder is small . . . and contains a small amount of bile which is inspissated. The cystic duct has apparently been occluded by large, hard, grey-blue nodes about the porta hepatis. The common duct is patent."

deposition. These changes can be noted in individual nodes on original roentgenograms over several years time. Different nodes demonstrate the various stages of the process.

Sections of the hilar nodes show a gradation in the changes from the earliest fibrosis (Fig. 10A) to large nodes in which the entire lymphoid structure is replaced (Fig. 10B). In the earliest lesions the capsule shows slight but distinct fibrosis but no calcification. The primary change is the replacement of the central portion

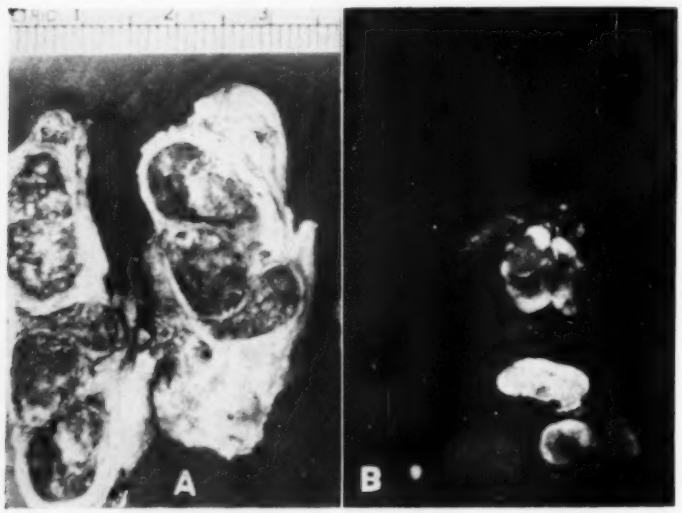
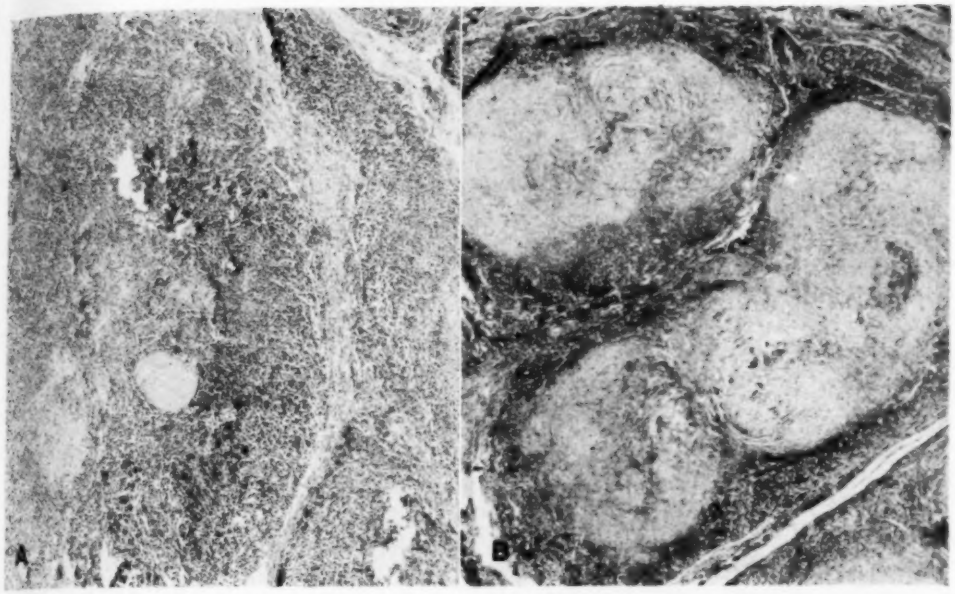


Fig. 9. A. Transected mediastinal nodes. B. Roentgenograms of smaller nodes immersed in water.

The heavier infiltrates in the right lung were bronchopneumonic consolidation histologically. Sections of lung and hilar nodes were obtained for our own studies. Silicotic changes involved all the lymph nodes described above, and the appearance does not vary from the histologic appearance in the other cases.

In addition to the tomograms taken to show the distribution of the calcium in previous instances, roentgenograms (Fig. 9B) of some of the smaller nodes were taken under water. Some showed earliest faint calcium density throughout the node, others beginning peripheral calcium deposit, and others extremely heavy and generally irregular, but *peripheral*, calcium

of the follicles by dense hyaline whorls of collagen. Fine particulate calcium is uniformly distributed. Silica crystals are seen by polarized light but are not very numerous. This process in its full development shows a dense fibrous capsule. The node structure is entirely replaced. There are numerous small nodules composed of a background of fibrous tissue, particulate calcium and, in some, clefts suggesting fatty acid crystals. These nodules lie in a dense fibrous stroma in which anthracotic pigment granules are present. In several nodes the more peripherally placed fibrous nodules show greater deposit than is seen in the center. Silica crystals are seen but again are not numer-



ous. One of the nodes examined shows the peripheral ring of calcium (Fig. 10C). This ring involves the inner portion of the dense fibrous capsule (9) predominantly, but extends irregularly into the fibrous substance of the node. None of the nodes shows any active process suggesting tuberculosis. None shows a homogeneous destruction and replacement by amorphous calcified material, such as is commonly seen in old calcified tuberculous nodes. The nodularity of the process is the striking difference. In the earlier phases, a fibrosis such as is seen in these nodes is not infrequently found in chronic infective lymphadenitis. However, it is not usual to find such early calcification in an otherwise uncomplicated infective lesion.

Sections of the lungs show cellular changes similar to those seen in the hilar lymph nodes. The nodules of fibrous tissues are well circumscribed and are of the same composition, with a uniform deposit of fine particulate calcium. *No annular deposit of calcium is seen.* Some alveolar walls show fibrous thickening. There are moderate emphysema and acute bronchopneumonia. No lesion indicative or suggestive of tuberculosis is seen.

Fig. 10. A. Early stage of silicotic adenopathy. B. Larger silicotic nodules with some calcium deposition in and about each nodule. C. Heavy subcapsular calcium deposit.

Portions of lung tissue and hilar nodes were inoculated into guinea-pigs, which remained healthy six weeks later. The animals were killed and examined. There

were no demonstrable lesions except at the site of injection. The local lesions were partially encapsulated by fibrous tissue and showed foreign-body reaction. Numerous crystals were visible in the encapsulated area. Some could be seen within phagocytes of multinuclear type. Eosinophilic cells were fairly numerous.

X-ray diffraction analysis[2] of the dry tissues shows over 1 per cent quartz and 20 to 25 per cent $Ca_3(PO_4)_2$. The acid insoluble residue contained 53 per cent quartz.

SUMMARY

The above studies of silicotic individuals, with regard to the unusual calcifications of a shell-like configuration, present a reasonable and conclusive hypothesis. These calcium deposits occur in silicotics as the result of silica without any superimposed tuberculous infection and probably no other infection. Silicotic material predominates, and the common denominator is *silicosis*. These calcifications occur long after the original exposure, especially in milder cases; they are not seen in severe cases terminating in death within a few years of exposure. The shell calcifications occur in previously existing lymph nodes but not in lung tissue. The calcium deposition begins diffusely throughout the node but later becomes more prominent beneath a heavy capsule that forms around the node.

Though the peculiar configuration of the calcium deposit seems to be characteristic of and unique to silicosis, it cannot be stated that it might not occur in chronic, non-caseating lymphadenitis of other origin. No other causes have been reported with any reasonable supporting evidence.

The presence of egg-shell calcifications in lymph nodes indicates silicosis.

1181 Cavanaugh Way
Sacramento, Calif.

[2] Courtesy of Donald Bailey, the Saranac Laboratory of the Edward L. Trudeau Foundation.

REFERENCES

1. DAVIES, T. W.: Silicosis in Slate Quarry Miners. Tubercle **20:** 543–555, 1939.

2. LOMMEL, F.: Über Beurteilung und Verhütung der Silikose (Staublungenkrankheit). Deutsche med. Wchnschr. **65:** 871–875, 1939.

3. REST, A.: The Roentgenogram and Some Chronic Non-tuberculous Pulmonary Conditions. Am. J. Roentgenol. **51:** 434–438, 1944.

4. RIEMER, A. D.: Egg-Shell Calcifications in Silicosis. Am. J. Roentgenol. **53:** 439–445, 1945.

5. RIGLER, L. G.: The Chest: A Handbook of Roentgen Diagnosis. Chicago, Year Book Publishers, 1946.

6. ROUVIÈRE, H.: Anatomie des lymphatiques de l'homme. Paris, Masson et Cie, 1932.

7. SCHULTE, G., AND HUSTEN, K.: Röntgenatlas der Staublungenkrankungen der Ruhrbegleute. Leipzig, G. Thieme, 1936.

8. SIEGAL, W., SMITH, A. R., AND GREENBURG, L.: Dust Hazard in Tremolite Talc Mining, Including Roentgenological Findings in Talc Workers. Am. J. Roentgenol. **49:** 11–29, 1943.

9. SWEANY, H. C.: Pathologic Interpretations of Roentgenologic Shadows in Pneumonoconiosis. J. A. M. A. **106:** 1959–1965, 1936.

10. SWEANY, H. C., PORSCHE, J. D., AND DOUGLASS, J. R.: Chemical and Pathologic Study of Pneumonoconiosis, with Special Emphasis on Silicosis and Silicotuberculosis. Arch. Path. **22:** 593–633, 1936.

SUMARIO

Calcificaciones Ovitestáceas en la Silicosis

Tratando de determinar la naturaleza y el significado de las calcificaciones a lo "cascarón de huevo" observadas, repasáronse 88 casos de silicosis con historias adecuadas, estudios radiológicos y, en algunos casos, hallazgos histológicos. Revistió interés particular un grupo de 8 casos en los que había calcificaciones de típica morfología, pero sin signos clínicos o histológicos de infección pulmonar tuberculosa o de otra naturaleza crónica.

El estudio indica que esos depósitos de calcio se presentan en los silicóticos por la acción de la sílice sin ninguna infección tuberculosa sobrepuesta. Las materias silíceas predominan, y el denominador común es la silicosis. Esas calcificaciones ocurren, sobre todo en los casos leves, mucho tiempo después de la exposición primitiva, sin que se observen en los casos graves que terminan en la muerte tras pocos anos de exposición. Las calcifica-

ciones ovitestáceas afectan los ganglios linfáticos, pero no el tejido pulmonar. El depósito de calcio comienza en forma difusa a través de todo el ganglio, pero luego cobra mayor relieve debajo de la gruesa cápsula que se forma alrededor del ganglio.

Aunque la peculiar configuración de los depósitos de calcio parece típica y propia de la silicosis, no cabe asegurar que no se presentaría en la linfadenitis no caseosa, crónica, debida a otras causas. Sin embargo, no habiéndose comunicado otras causas con razonables datos confirmatorios, cabe decir que la presencia de calcificaciones en cascarón de huevo en los ganglios linfáticos indica silicosis.

DISCUSSION

Philip J. Hodes, M.D. (Philadelphia, Penna.): Drs. Grayson and Blumenfeld have been talking on an extremely controversial subject, about which little is known. Because of this, their paper, based as it is upon histopathologic observations, is a timely and an important one. They are to be congratulated upon the objectivity with which they have approached the problem and the clarity with which it was presented. Whereas we agree that tuberculosis may play no part in the production of these egg-shell calcifications, we are somewhat critical of their conclusion that "these calcium deposits occur in silicotics as the result of silica." Time and experience may prove them right, and there is no question but that others do agree with them.

As we reviewed the authors' paper and excellent exhibit, we found ourselves wrestling with the following thoughts.

Why, we asked ourselves, did three men who came to autopsy with egg-shell calcifications show these remarkable changes when they had worked for only two years as grinders, twenty, twenty-two, and thirty-four years previously? True it was that silica was found in their lungs at autopsy—but did this mean they had silicosis and that the silica had excited the formation of these egg-shell calcifications? We found it difficult to conclude that because the patient had silica in the lungs, and because egg-shell calcifications were present, the two were unequivocally related as cause and effect.

We were amazed to find the large egg-shell calcifications in a supraclavicular fossa in one of the films in the exhibit—a unique finding in our experience with silicosis. We were also worried by the large disseminated calcified nodules in the lung parenchyma of some of these patients which did not seem to fit the usual picture of silicosis. And as we drew upon our own experiences with silicosis and found it difficult to remember even a few patients with egg-shell calcifications, we wondered why it was that here, in California, the authors were able to find 40 patients with egg-shell calcifications in a group of 200 silicotics, an incidence of 20 per cent.

Furthermore, we were reminded of Davies' experience with a large group of slate-quarry miners, in whom he found large numbers of egg-shell calcifications, though he failed to find any in silicotics from a neighboring mine.

The answer may lie in the authors' histopathologic sections. In discussing their microscopic findings in these egg-shell calcifications they state that "in the earlier phases a fibrosis, such as seen in these nodes, is not infrequently found in chronic infective lymphadenitis"!! How then do the authors rule out infection as a possible factor? Why couldn't some bizarre infection such as histoplasmosis, toxoplasmosis, or some as yet unknown infective agent, endemic in the city county, or state in which the patient lived, be responsible for these changes? Why, too, couldn't it be a chemical agent other than silica that is to blame? Could the agent be in the food or the water the patient drinks? One cannot ignore the fact that, whereas many different occupations have been incriminated, there have been many people in similar industries in other parts of the country who have remained unaffected.

We wish to re-emphasize that Dr. Grayson and Dr. Blumenfeld have given us important observations which add to our knowledge of egg-shell calcifications. It has been a pleasure to read and worry over their paper and a privilege to discuss it; that we may see in its contents different implications is but added testimony of the uncertainty that has characterized the literature in the past.

Moreton J. Thorpe, M.D. (Reno, Nevada): I think that Dr. Grayson and Dr. Blumenfeld are particularly to be congratulated on the clarity with which they have presented the pathologic slides and almost definite proof of the fact that the calcifications which they describe are due to silica. I have nothing to add except for the sake of emphasis; I would like to mention that I think that these cases are not uncommon. We have seen many of them in Nevada, where I believe that silicosis is possibly more common than it is here in California, as quartz predominates in the mines.

We have seen calcifications almost the size of clusters of seedless grapes, and the particular thing about them is that they are symmetrical, bilateral, and evenly distributed about both hili.

As Dr. Grayson mentioned, they are most commonly seen, or practically always seen, in cases which are uncomplicated by tuberculosis and in which the silicosis is not pronounced. Those patients who come down with a severe silicosis have a marked bronchitis, and if there is a complicating tuberculosis, they are apt to die before silicosis advances to the stage of calcification.

I remember that years ago there were several questions as to whether silicosis did calcify, as it had been thought that tuberculosis was one of the few conditions that was ever subject to calcification. I think that it can now be pretty well certified that calcification occurs in histoplasmosis, in aspergillosis, in coccidioidomycosis, in tuberculosis, in silicosis, and, of course, in pleural hemorrhages; but the calcifications under consideration are very apparent, particularly in Nevada, as we see them in quartz miners without a complicating tuberculosis so far as the x-ray findings are concerned.

Dr. Grayson (*closing*): I shall try to answer some of the questions that Dr. Hodes has propounded.

Why did some of these men have only two years of exposure? We have no accurate measurements of dust particles, the number inhaled, etc. I can only say that short exposure may produce silicosis. One of the men that was a grinder was told by his predecessor that he should not stay on the job for more than one year, because every man who stayed longer than that died a few years afterward.

As far as nodes in the supraclavicular region are concerned, it was interesting to note that in our last case the patient had complete obstruction of the cystic duct of the gallbladder by silicotic nodes. Silicosis has been reported in the spleen; silicosis has been reported in the pericardium, and there is no reason to doubt that silicosis doesn't extend to the supraclavicular nodes.

Why do we find so many of these silicotics here? It is probably partly due to the conditions in the western part of the United States, where we have so many prospectors who are exposed to a minor degree. Of course, they are out in the open. They have a mild silicosis and are not exposed to certain infections, such as tuberculosis, which is more prevalent under crowded conditions and which has been reported in the Welsh miners as well as in the German miners.

Dr. Hodes asked a question about other types of infection, since we made the statement that the fibrosis is similar to that in infectious lymphadenitis. I don't know that I can answer his question very well, except to say that all we have is a known non-specific reaction which appears as fibrosis. It may occur in any sort of condition which incites inflammatory changes. The important point that we wish to bring out is that peculiar calcifications demonstrable on the chest roentgenograms do give us a hint that this is silicosis rather than any other type of inflammatory disease, because silicosis is the common denominator. We have looked for these calcifications in thousands of chest roentgenograms which include the chest films that we read from day to day, the chest films in the tuberculosis sanatoriums, the chest films in the various surveys, etc. Only last week I found one film which looked something like these in a four-year-old girl who was known to have tuberculous adenitis. Her mother had active pulmonary tuberculosis, and the girl was in the healing stage. We observed her for several years and had noted that the calcifications became solid.

Something should be said, I think, about the fact that Dr. Hodes feels that the calcification might be due to some local condition. These patients come from all over the world. One of these men was a glass blower who worked in the pouring section of the glass factory in continental Europe during his apprenticeship. He had no exposure to silica dust thereafter. One of the men had worked on the Panama Canal and in the East as a tunnel worker. The axe grinders were in the East.

Now we know that silica can be demonstrated in the lungs of farmers from areas where the silica content of the topsoil is high. However, those men do not develop silicosis which can be determined clinically or radiographically, whereas all of these others had clinical and radiographic evidence of silicosis. Therefore, it seems to be a matter of degree rather than of any of the other factors.

Intrathoracic Goiter

Its Incidence, Symptomatology, and Roentgen Diagnosis[1]

JAMES J. McCORT, M.D.

Boston, Mass.

OVER A PERIOD OF six and a half years a total of 908 thyroidectomies have been performed at the Massachusetts General Hospital. In 28, or 3.1 per cent, the thyroid was proved to be intrathoracic. In arriving at this figure, the classification of Wakeley and Mulvany (50) was followed, that is, only those cases in which the major portion of the goiter lay within the thorax were considered as intrathoracic. This criterion has also been recommended by Means (38). A classification determined by the relation of the goiter to the aortic arch is less satisfactory, since the position of that structure varies with the individual and with the shape of the chest. In 20 of the 28 intrathoracic goiters comprising the present series, the major portion of an enlarged thyroid extended into the thorax; in 8, the entire goiter lay within the thorax; all extended to or beyond the aortic arch.

The incidence of intrathoracic goiter reported in the literature shows some variation because there are no uniformly accepted criteria for its classification. Lahey and Swinton (33), counting only those goiters which extended to or beyond the arch of the aorta, found an incidence of 7 per cent in 5,131 thyroidectomies, while von Zweigbergk (52), using the same criteria, reported 0.8 per cent in 2,625 such operations. Joyce (27), who included both substernal and intrathoracic goiters in his study, found that they represented 12.9 per cent of 1,334 thyroidectomies. It is interesting to compare with these figures those of Kirshbaum and Rosenblum (29), who discovered among 1,222 nodose goiters at autopsy only 3, or 0.25 per cent, that were intrathoracic. In other large series of thyroidectomies (4, 11 28, 50) the incidence of intrathoracic goiter ranges from 1 to 10 per cent. The relatively high percentage in the present series may be weighted somewhat by that fact that the cases were drawn from an active thoracic surgical service.

CLINICAL DATA

The ages of the 28 patients in the group under investigation (14 male and 14 female) ranged from thirty-five to seventy-five years; only 3 were under forty, and 20, or 71 per cent, were over fifty years old. This latter point is important in the differential diagnosis and agrees with the known tendency of nodular goiter to occur most commonly in persons over fifty (43).

As might be expected, the most frequent complaint, noted in 10 patients, was swelling of the neck. The onset of the swelling in all patients was gradual, occurring over a period of several years (Table I). Dyspnea on exertion was the second most common complaint. Eight patients had chronic cough, described as irritating and brassy; except in one patient who had demonstrable bronchiectasis, it was nonproductive. Dysphagia was only an occasional complaint and none of the 4 patients experiencing it exhibited signs of cachexia. Two important but infrequent symptoms were dyspnea on lying down and a choking sensation. Characteristically these two symptoms are worse when the head is turned toward the side on which the goiter lies; immediate relief usually occurs on assumption of the upright position. In 4 of the group there were no symptoms, the goiter being an incidental finding; 2 were found accidentally in

[1] From the Department of Radiology, Massachusetts General Hospital, Boston, Mass. Accepted for publication in July 1948.

TABLE I: CLINICAL DATA FOR TWENTY-EIGHT INTRATHORACIC GOITERS

Symptoms		Physical Findings		Laboratory Findings		
Swelling of neck	10	Enlargement of thyroid	20	B.M.R.	Within normal limits	15
Dyspnea on exertion	9	Deviation of trachea	4		Elevated	3
Cough	8	Dilatation of neck veins	3			
Dysphagia	4	Disappearing mass in neck	1	ECG	Within normal limits	6
Dyspnea lying down	4	Vocal cord paralysis	1		Abnormal	6
Choking sensation	2	Left diaphragm paralysis	1		Coronary heart disease	2
Hoarseness	3	Apical systolic murmur	1		Left ventricular strain	1
Backache	2				Right blundle branch block	1
Nervousness	2				Premature beats	1
Fatigability	2				Unusual rotation heart	1
Weight loss	1			Protein bound I_2		
Precordial pain	1				Normal	3
Mid-sternal crampy pain	1				Elevated	1
Pain and weakness of right arm	1					
No symptoms	4					

patients hospitalized for unrelated disease and 2 through photoroentgen survey. It is of interest that 3 of the patients gave a history of previous thyroidectomy for nodular goiter, none of recent date. It is not possible to state whether in these instances the intrathoracic goiter developed subsequently or whether it was present but undiscovered at the first operation.

On physical examination (Table I), some degree of enlargement of the thyroid was noted in 20 of the 28 patients. In most instances the goiter moved upward with deglutition, and it was not possible to palpate its lower pole. Pape (40) has pointed out that the demonstration of even a very small goiter in the neck, showing this diagnostic feature, is highly significant. Coupled with the roentgen finding of a superior mediastinal tumor, it strongly suggests an intrathoracic goiter. Careful palpation of the neck should therefore never be omitted in the study of an undiagnosed mediastinal tumor.

Deviation of the trachea was not found as frequently on physical examination as on roentgen study, which will be discussed later. Dilatation of the neck veins in 3 patients indicated pressure on the jugular veins. In only one patient was a *goitre plongeant* demonstrated. This is the type, first described by Malard (35) in 1879, which disappears within the thoracic cavity only to bob up into the neck when the patient coughs or swallows. It is said to be accompanied by much more subjective change than other types of goiter. In a series of 1,300 thyroidectomies, Wakeley (50) recognized only 2 cases of *goitre plongeant*. Some authors unfortunately use this term to describe all goiters which extend into the thorax.

Vocal cord paralysis, due to pressure on the recurrent laryngeal nerve, was observed in only one instance; function of the cord did not return postoperatively. In a somewhat similar case, reported by Parsons (41), vocal cord function returned to normal following operation. Waugh (51), on the basis of a large number of routine preoperative laryngeal examinations, has estimated that paralysis of a vocal cord due to pressure occurs in 10 per cent of all patients with goiter. One patient in the present series had unexplained paralysis of one leaf of the diaphragm; this may have been an incidental finding.

Only 3 of the 28 patients showed evidence of hyperactivity of the thyroid; in one, the toxic state was described as "subclinical," and in the other 2 it was mild. The basal metabolic rate was elevated in these 3 patients (Table I) but was within normal limits in the remaining 15 on whom the test was performed. The electrocardiogram was abnormal in 6 cases, but since the majority of the series were in the older age group such changes were to be expected, as in any similar group. In none were the abnormalities considered severe enough to contraindicate surgical

intervention. Determination of the protein-bound iodine of the blood was made in 4 patients; in only one instance was it found to be elevated above the accepted normal (3.5 to 7.5 mg. per cent), and this patient showed other evidence of mild hyperactivity of the thyroid.

In none of the 28 patients were there any preoperative or postoperative complications of any severity. A review of the literature would indicate, however, that complications can and do occur in association with intrathoracic goiter. Colp (10) and Fiessinger *et al.* (17) reported cases in which emergency operation was required for acute dyspnea due to tracheal compression. Recurring pleural effusion accompanying a benign intrathoracic goiter has also been recorded (5, 30). That intrathoracic goiter may precipitate a laryngeal crisis in a patient with tabes dorsalis has been suggested by Holsti (24); anginal pain, disappearing after thyroidectomy, has been reported by Edeiken and Rose (15). Chylothorax due to compression of the thoracic duct was described by Schultze (44), and thyroiditis due to extension from a lobar pneumonia was reported by Kirshbaum and Rosenblum (29).

PATHOLOGIC ANATOMY

The majority of the removed thyroid glands were nodular, either grossly or microscopically. Involution and/or hyperinvolution, occasionally associated with hyperplasia, were the most frequent findings (Table II). Toxic diffuse hyperplasia, however, was not seen; it is exceedingly rare for that type of goiter to extend into the thorax to an appreciable degree (8, 31). In only one of the 28 cases was carcinoma found; this is essentially within the expected incidence of malignancy in nodular goiter (9). There is no evidence to indicate that the percentage of malignant tumors is higher in intrathoracic than in other goiters.

TABLE II. HISTOPATHOLOGIC CHANGES IN CASES OF INTRATHORACIC GOITER

Involution	12
Hyperinvolution	8
Hyperplasia and involution	4
Fetal adenoma	2
Carcinoma	1

All of the intrathoracic goiters in the group had their origin in a normally situated gland in the neck. This was checked at operation, and in all cases the continuity of the intrathoracic tumor with the gland in the neck could be demonstrated, although in a few instances the connection consisted of only a fibrous band or vascular pedicle. In no instance was there evidence that the goiter arose in ectopic thyroid tissue already present in the mediastinum as the result of a developmental arrest. This appears to be a very unusual occurrence; only a few such cases are recorded in the literature (16, 36, 42, 46). (Cases in which thyroid tissue was found in a mediastinal teratoma were excluded from this study.)

The mechanism by which a cervical goiter enters the thorax has been ably expounded by Judd (28) and by Lahey (33). These authors point out that the act of deglutition causes the goiter to move up and down in the thoracic inlet. Since posterior growth is prevented by the cervical spine, and anterior and lateral growth is hindered by the strap muscles—omohyoid, sternocleidomastoid, sternohyoid and sternothyroid—the enlarging gland in a small percentage of cases will extend farther and farther downward through the thoracic inlet. Eventually it becomes incarcerated within the mediastinum and can no longer return to its original position. Further growth, which is slow and gradual over a period of years, will then take place within the mediastinum, where the loose areolar tissue offers little resistance.

Table III shows the anatomical location of the goiter within the mediastinum. It will be seen that the greater number were found anterior or anterolateral to the trachea. They were located on either side with almost equal frequency and in some cases partially surrounded the trachea; occasionally they were bilateral with a result-

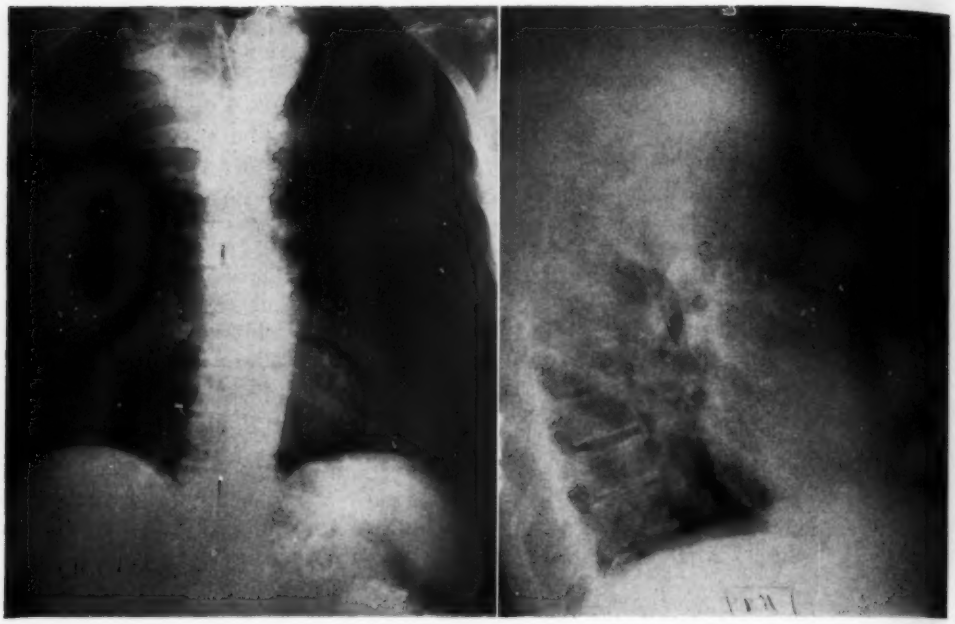


Fig. 1. A. A., a 63-year-old white male, complained only of pain and weakness in the right arm. The only finding on physical examination was that the trachea was deviated to the right. The patient had received a therapeutic trial of radiation therapy at another institution, with some decrease in the size of the tumor. The goiter was found to lie posterior to the trachea and anterior to the esophagus. It was slightly nodular and moved with swallowing. Due to its location, it was removed by a transthoracic approach.

ing "S" shaped curve of the trachea. The goiter was situated between the trachea and the esophagus in 6 cases (Fig. 1), and posterior to the esophagus in 3 (Fig. 2).

TABLE III: LOCATION OF GOITER WITHIN THE THORAX
(All 28 goiters were in the superior mediastinum)

Anterior to trachea		4
Anterolateral to trachea		12
Right	5	
Left	7	
Bilateral		3
Between trachea and esophagus		6
Posterior to esophagus		3

The reason for the intrathoracic goiter assuming an anterior or posterior position is conjectural. Goiters arising from the inferior poles and from the isthmus tend to lie anteriorly, usually anterior to the carotid and subclavian arteries. If the origin is in the lateral portion of the lobe, the goiter as a rule lies posterior to the trachea and esophagus, and posterior to the arterial trunks. Whether the carotid and subclavian arteries influence the direction of the descent of the goiter is speculative, but it remains a possibility.

ROENTGEN FINDINGS

Roentgenoscopy and roentgenography contributed the most important information toward diagnosis in this series. Under the fluoroscope, the tumor was studied for motion with deglutition, for the presence or absence of pulsation, for change in shape with change of the patient's position, and for its relationship to the other structures of the superior mediastinum. Experience proved that films should include the entire trachea up to the larynx, and that the lateral projection is best made with the patient's arms extended posteriorly. A good outline of the esophagus will be obtained if a thick barium paste is swallowed immediately before exposure of the film.

The intrathoracic thyroid in all 28 cases was located in the superior mediastinum. Only 2 cases have been found in the

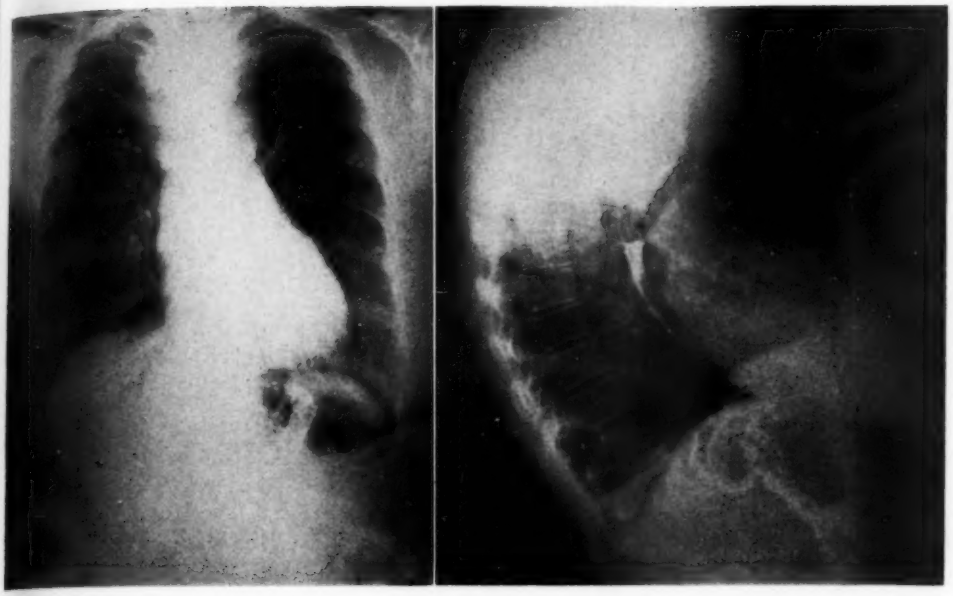


Fig. 2. I. E., a 56-year-old white female, complained only of chronic cough. One year previously a diagnosis of mediastinal tumor had been made at another hospital. For this x-ray therapy had been given, directed to the mediastinum, without any subsequent change in the size of the tumor. On entering this hospital the physical examination was essentially negative. The thyroid was not palpable. An electrocardiogram showed no abnormality. The roentgenogram showed a slightly nodular tumor, which was on the left and posterior to the trachea and esophagus. Displacement of the trachea extended to the level of C-7. The tumor showed no calcification, but there was a pleural reflection, and the mass moved upward with swallowing. At operation, the goiter was seen to arise from the left lobe of the thyroid. It was completely removed through a cervical incision, by Dr. E. D. Churchill.

medical literature in which the position was lower than this (6, 47).

One of the earliest clinical observations on intrathoracic goiter, first mentioned by Bonnet (3) in 1851, was that it caused a deformity of the trachea. In its descent into the thorax, the goiter will invariably displace and sometimes compress the trachea. Minor degrees of lateral or anteroposterior displacement are difficult to determine by palpation alone. This is particularly true when the patient has a short neck and a thick wide chest, which is the common genotype in this condition (4). Tracheal displacement was readily determined by roentgenography using the technics described. For this reason, the films were taken with the patient erect, with the spine as straight as possible, and with the chin up. Tracheal displacement was evident in 27 of the 28 patients, the amount of displacement varying with the size of the tumor and with its location. Maximal displacement occurred opposite the greatest diameter of the tumor.

A most valuable diagnostic point, found in 23 of the patients, was that the deviation of the trachea began high in the neck, often at the larynx (Fig. 3). This high displacement resulted at times in an angulation of the larynx away from the side containing the goiter; it was best demonstrated when the hypopharynx was outlined by barium mixture. In the study of superior mediastinal tumors, therefore, displacement of the upper trachea is an important differential point (1). Dermoids, teratomas, bronchiogenic cysts and aneurysms of the arch of the aorta or of the innominate artery, cause displacement of the trachea, but not to such a high level. The same statement applies to tumors composed of enlarged paratracheal lymph nodes due to malignant lymphoma or sarcoid in which the nodes are the only visible manifestations of the

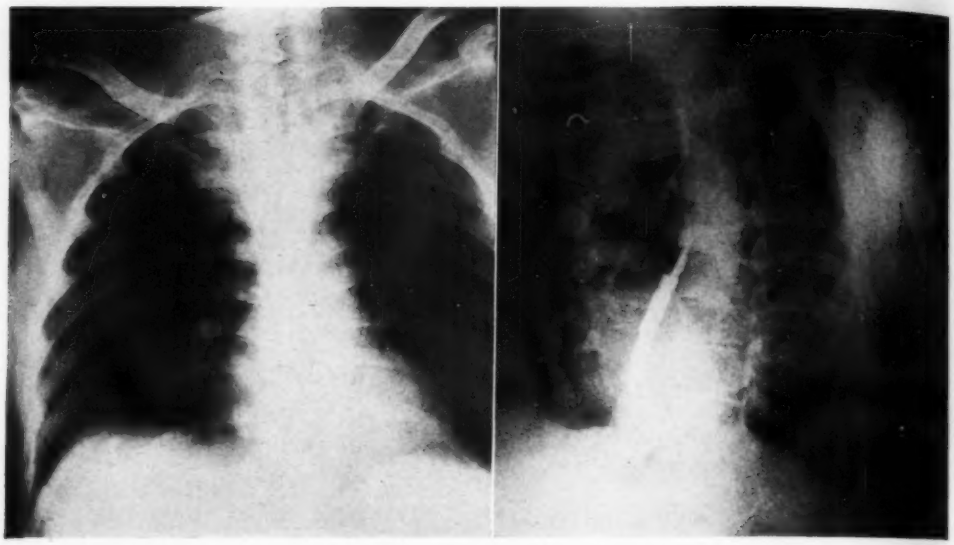


Fig. 3. A. A., a 64-year-old white male, entered the hospital complaining of slight dysphagia, chronic cough, and a choking sensation when he lay down. Physical examination showed the veins in the neck to be distended, but the thyroid was not palpable. An electrocardiogram showed no definite abnormalities. In this patient the goiter was in the right anterior portion of the superior mediastinum. It displaced the trachea and esophagus posteriorly and to the left. The outline of the tumor is smooth and it contains no calcification. There was marked upward motion with swallowing and a pleural reflection.

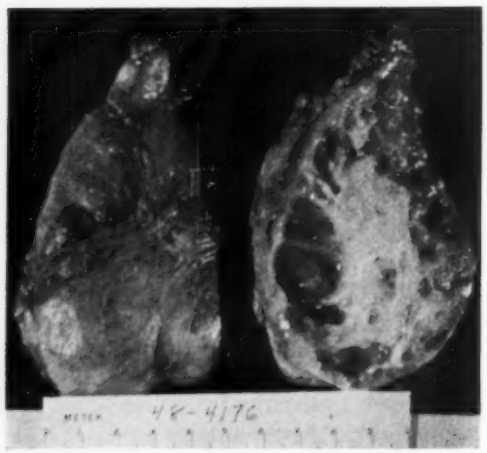


Fig. 3B. Resected specimen (from case shown in Fig. 3) which was removed through a low cervical incision by Dr. E. D. Churchill. It was attached to the right lobe of the thyroid. The gross nodularity is apparent, although not demonstrable on the roentgenograms. Microscopically, involution and hyperinvolution were observed.

disease. After removal of the intrathoracic goiter, the trachea resumed its normal anatomical position—a fact also noted by Cattell and Hare (7) and Curtis (13).

Slight compression of the trachea occurred in all cases when there was displacement of this structure; severe compression was an infrequent finding. It was seen where the goiter was bilateral or where it partially surrounded the trachea. This compression can explain the complaint of stridor and difficulty in breathing when the patient is lying down.

With displacement of the trachea, there was usually a corresponding displacement of the esophagus. When the goiter was interposed between the trachea and the esophagus, these structures were separated. In the group presented, there was no fixation of the esophagus to the goiter, and the esophageal mucous membrane was always intact; no obstruction or delay in the passage of barium through the esophagus was noted.

It is important to determine by roentgenoscopy whether the tumor in the superior mediastinum moves with swallowing. This observation was made in 19 of the series: the tumor moved with deglutition in 16, or 84 per cent; in only 3 was no

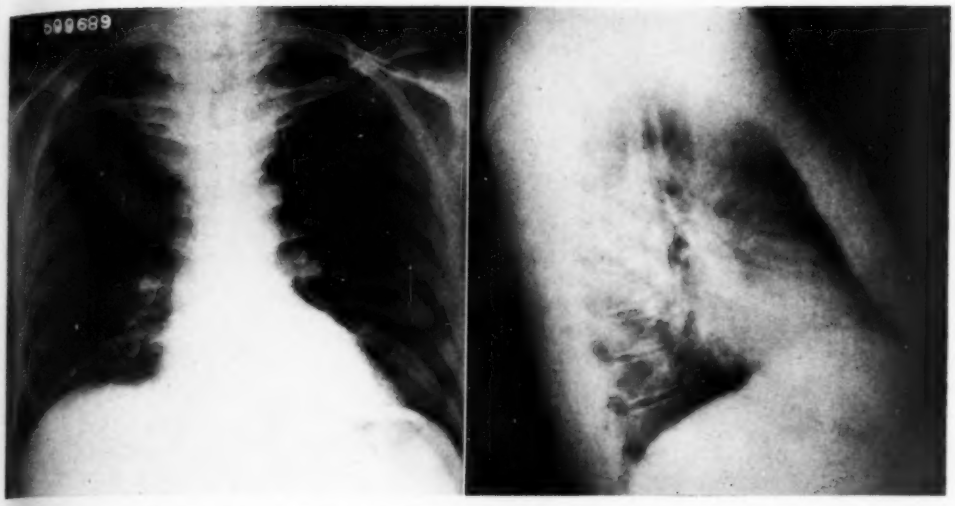


Fig. 4. R. M., a 56-year-old white male, entered the hospital for a physical check-up and regulation of a mild diabetes. The goiter was an incidental finding. Physical examination disclosed a slight dilatation of the neck veins and a small palpable mass in the right neck. A chest roentgenogram brought to light a mass in the anterior superior mediastinum, which displaced the trachea posteriorly and to the left. It was slightly nodular and showed no calcification. A reflection of the mediastinal pleura below the goiter at the base of the heart is seen. This goiter did not move with swallowing, and at operation the surgeon, Dr. Richard H. Sweet, found it to be tightly wedged beneath the sternum. It was removed through a cervical incision.

motion reported. In one of the 3, the surgeon found at operation that the goiter in the thorax was attached to the gland in the neck by only a thin band of connective tissue. This may not have been of sufficient strength to elevate the goiter upon swallowing. In a second, the entire goiter was discovered to be tightly wedged beneath the sternum (Fig. 4).

The upward motion of the intrathoracic goiter with deglutition can be explained as follows. Since it is a downward extension from a normally situated gland, which is firmly attached by the thyroid cartilage to the hypopharynx in the neck, the goiter, unless restrained, must move upward with closure of the glottis. This movement, when present, is marked. Other superior mediastinal tumors, such as bronchiogenic cysts, enlarged paratracheal lymph nodes, and occasionally but rarely aortic aneurysms which are attached to the trachea by inflammatory changes, may move with swallowing, but the motion, which is less pronounced, is due to attachment of the tumor to the trachea. Failure of an intrathoracic goiter to move with swallowing does not necessarily indicate malignant change; in the one case of carcinoma in this series movement of the goiter was observed when the patient swallowed.

It is useful also to observe the tumor fluoroscopically for pulsation; it is essential if aneurysm is considered in the differential diagnosis. Not all aneurysms pulsate, however, and on the other hand an intrathoracic goiter may show a transmitted pulsation, so that it is often necessary to invoke other criteria to make this differentiation. Aneurysm may be excluded if the tumor can be seen separate from the aortic arch on roentgenoscopy. Where the two structures overlap, the goiter will make a sharp, sometimes acute angle with the aortic arch (12, 39), while an aneurysm will tend to follow the direction and curve of the normal arch. If the equipment is available, angiocardiography offers the most certain way of excluding aneurysm, particularly if involvement of the innominate artery is suspected.

Calcification of part of the intrathoracic goiter was present in 7 of 28 cases, or in

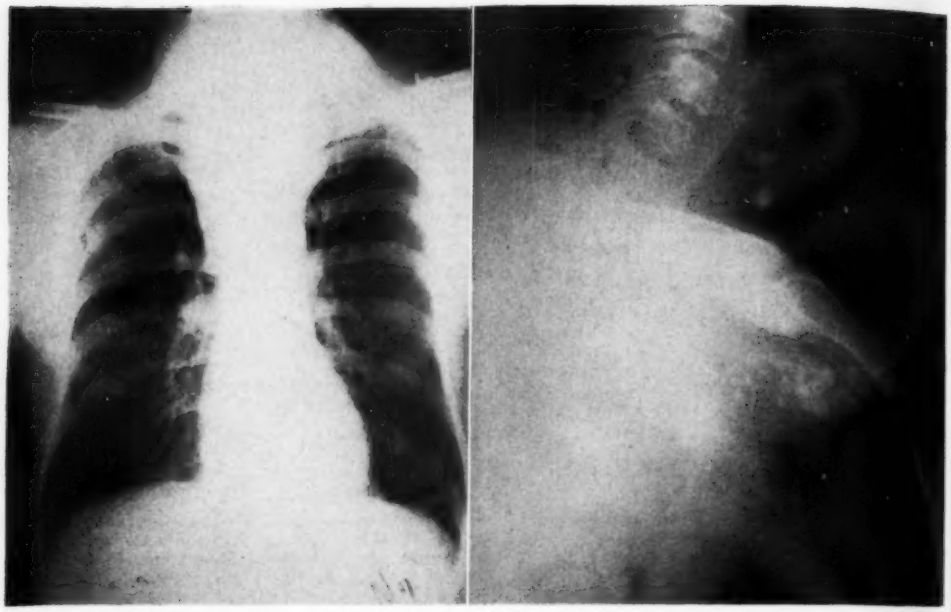


Fig. 5. P. C., a 45-year-old white male, complained of swelling of the neck, dyspnea, cough, and weight loss. Physical examination revealed a large hard mass in the neck. The basal metabolic rate was not elevated. The goiter was nodular and anterior to the trachea, being present on both sides of that structure. It showed a heavy, dense, and flocculent calcification and moved upward with swallowing. Extirpation was done through a cervical incision, and carcinoma of the thyroid was found.

25 per cent. The calcification, when it occurred, was soft, amorphous, and somewhat irregular in outline, and lay within the body of the tumor; one exception was noted, in which the calcification was ring-like and lay in the wall of the goiter. The one carcinomatous goiter was heavily calcified (Fig. 5).

Grossly, the majority of the goiters were found on surgical removal to be nodular. It was surprising, therefore, in a restudy of the films to find fairly smooth outlines (Fig. 3). In only 5 of the 28 cases could nodularity be demonstrated, and in these it was not marked. This discrepancy between the morphologic appearance of the tumor and its roentgen outline may be explained by two factors. In the first place, the goiter within the superior mediastinum tends to displace the venous channels laterally, so that in some cases the lateral margin as seen in the roentgenogram is formed by these veins. In the second place, the conventional roentgenogram is taken with the patient erect, and in this position the goiter hangs suspended in the superior mediastinum, since in the uncomplicated case its only attachment is in the neck. In a soft tumor this may cause a smoothing-out of the margins, similar to that which may be seen when a partially filled paper sack is suspended by its neck.

One other observation of diagnostic importance is that there was almost always a reflection of the mediastinal pleura below the tumor (Fig. 4). This was demonstrable where the mediastinal pleura united with the pericardium at the base of the heart and indicated the mediastinal, rather than the pulmonary, location of the tumor.

The presence of the goiter within the chest must result in some pressure atelectasis in the adjoining lung; this will vary directly with the size of the goiter. Gross atelectasis or bronchial occlusion was not noticed in any patient.

Ivanissevich *et al.* (26) have found

tomography to be of value in showing the continuity of a goiter in the thorax with the goiter in the neck. This type of examination should be of help in delineating the partially intrathoracic goiter but not the completely intrathoracic one.

TREATMENT

There is only one satisfactory treatment for intrathoracic goiter, and that is surgical removal. This is indicated for several reasons: 7.2 per cent of nodular goiters are said to be malignant (9); sudden tracheal compression may result in asphyxiation (this is possible if there is hemorrhage into the gland or if growth is sudden and rapid); interference with the venous return from the head may result in syncope (22). The technic of surgical operation has been described in numerous articles (2, 8, 11, 14, 18–20, 23, 25, 31, 32, 34, 37, 45, 48).

There is no indication for deep x-ray therapy of the intrathoracic thyroid. A nodular goiter may decrease in size, but only after a very heavy dose of radiation; or it may not be affected at all. Two patients in this series were given radiation therapy at other hospitals before coming to operation, but details as to dosage and technic are lacking. With the rapid advances made in thoracic surgery during recent years, the use of a therapeutic test dose of radiation applied to an undiagnosed mediastinal tumor should be discarded. The information it furnishes is often unreliable, and the possibility of doing harm to normal lung tissue in the path of the beam is very real. Watchful waiting is also a questionable procedure, as the opportunity for successful resection may be lost during the waiting period. Surgical exploration is always indicated when clinical and radiologic investigation fails to reveal the nature of a mediastinal mass.

SUMMARY AND CONCLUSIONS

1. In a series of 908 thyroidectomies for all causes, a partially intrathoracic goiter was found in 20 patients and a completely intrathoracic goiter in 8.

2. Twenty-four of these 28 goiters were non-toxic and nodular, 3 were nodular with a mild degree of hyperactivity, and 1 showed carcinoma. There were no instances of toxic diffuse hyperplasia with exophthalmos.

3. The patients were in the older age group, the majority being over fifty. There was no sex predominance.

4. The most constant complaints, in order of frequency, were swelling of the neck, dyspnea on exertion, cough, dysphagia, dyspnea on lying down, a choking sensation, and hoarseness. Four patients were asymptomatic.

5. On physical examination, the most common and most important finding was a palpable enlargement of the thyroid; this was found in all patients in whom the intrathoracic extension was partial. Deviation of the trachea and dilatation of the neck veins were not often noted. One patient showed a *goitre plongeant* and one a vocal cord paralysis.

6. All 28 goiters were located in the superior mediastinum; 19 were anterolateral to the trachea, 6 behind the trachea, and 3 behind the esophagus.

7. The significant roentgen findings were as follows:

(*a*) Displacement of the trachea by the mass, present in 27 of 28 cases.
(*b*) Displacement of the trachea beginning high in the neck, frequently at the larynx and with some tilting of the larynx.
(*c*) Compression of the trachea, often present but not in marked degree.
(*d*) Displacement or compression of the esophagus accompanying similar changes in the trachea.
(*e*) Upward motion of the goiter with swallowing, observed in 84 per cent of the patients examined.
(*f*) Calcification within the goiter, noted in 25 per cent of the cases.
(*g*) A smooth or only slightly nodular outline of the tumor.
(*h*) Reflection of the mediastinal pleura below the goiter.

8. Complete surgical removal was accomplished without complication in all the cases; this is the treatment of choice for any intrathoracic goiter.

Department of Radiology
Massachusetts General Hospital
Boston 14, Mass.

BIBLIOGRAPHY

1. ADAMS, R.: In discussion of Cabot Case 28412: Bronchiogenic Cyst Arising from Trachea. New England J. Med. **227:** 560–563, 1942.

2. BLAIN, A. W., AND DEMATTEIS, A.: Surgical Management of Substernal and Intrathoracic Goiter. Am. J. Surg. **69:** 160–167, 1945.

3. BONNET, B.: Mémoire sur les goitres que compriment et déforment la trachea. Gaz. méd. de Paris, 1851, p. 772.

4. BRITTO, R.: Bocios intratorácicos, Hospital, Rio de Janiero **29:** 341–382, 1946.

5. Cabot Case 24191: Substernal Colloid Goitre with Compression of Innominate Veins. New England J. Med. **218:** 816–819, 1938.

6. Cabot Case 22352: Mediastinal Goiter. New England J. Med. **215:** 403–405, 1936.

7. CATTELL, R. B., AND HARE, H. F.: Position of Trachea before and after Removal of Substernal Goiter. S. Clin. North America **23:** 781–792, 1943.

8. CLUTE, H. M., AND LAWRENCE, K. B.: Intrathoracic Goiter. Am. J. Surg. **54:** 151–160, 1941.

9. COLE, W. H., SLAUGHTER, D. P., AND ROSSITER, L. J.: Potential Dangers of Nontoxic Nodular Goiter. J. A. M. A. **127:** 883–887, 1945.

10. COLP, R.: Substernal Goitre with Acute Dyspnea. Ann. Surg. **97:** 280–281, 1933.

11. CRILE, G., JR.: Intrathoracic Goiter. Cleveland Clin. Quart. **6:** 313–322, 1939.

12. CROTTI, A.: Thyroid and Thymus. Philadelphia, Lea & Febiger, 1918. Quoted by Higgins (21)

13. CURTIS, G. M.: Intrathoracic Goiter. J. A. M. A. **96:** 737–741, 1931.

14. DECOURCY, J. L., AND PRICE, C. A.: Intrathoracic Goiter. Am. J. Surg. **64:** 257–262, 1944.

15. EDEIKEN, J., AND ROSE, E.: Relief of Anginoid Pain Following Removal of Intrathoracic Non-toxic Nodular Goiter. Am. J. M. Sc. **196:** 395–400, 1938.

16. EGGERS, C.: Discussion of paper by Parsons, W. B. (41).

17. FIESSINGER, N., WELTI, H., DUPUY, R., AND CASTAIGNE, P.: Compression médiastinale dramatique due à un goitre intra-thoracique annulaire. Thyroidectomie d'urgence Guérison. Bull. et mém. Soc. méd. de hôp. de Paris **61:** 32–34, 1945.

18. FURBER, T. M.: Local Anæsthesia for Retrosternal Goitre. M. J. Australia **2:** 998–999, 1938.

19. VON HABERER, H.: Struma retromediastinalis. Zentralbl. f. Chir. **65:** 906–910, 1938.

20. HICKEN, N. F.: Recognition and Management of Intrathoracic Goiters. Nebraska M. J. **21:** 41–48, 1936.

21. HIGGINS, C. C.: Intrathoracic Goiter. Arch. Surg. **15:** 895–912, 1927.

22. HINSHAW, H. C., AND RUTLEDGE, D. I.: Lesions in Superior Mediastinum Which Interfere with Venous Circulation. J. Lab. & Clin. Med. **27:** 908–916, 1942.

23. HOLLENBERG, H. G.: Intrathoracic Goiter. Report of Patient Operated on Through Thorax. J Thoracic Surg. **15:** 283–289, 1946.

24. HOLSTI, O.: Lowseated Goiter as Chief Provoker of Laryngeal Crises in a Tabetic Person. Acta med. Scandinav., supp. 170, 1946, pp. 92–98.

25. HUNT, C. J.: Technical Problems in Surgical Management of Large Cervical and Intrathoracic Goiter. West. J. Surg. **48:** 524–528, 1940.

26. IVANISSEVICH, O., FERRARI, R. C., AND RIVAS, C. I.: Un signo de certeza para el diagnóstico de los bocios sumergidos. Bol. y trab. de la Soc. de cir. de Buenos Aires **23:** 281–283, 1939.

27. JOYCE, T. M.: Incidence of Substernal and Intrathoracic Goiters. Arch. Surg. **41:** 364–369, 1940.

28. JUDD, E. S.: Intrathoracic Goiter. Internat. Clin. **1:** 149–157, 1920.

29. KIRSHBAUM, J. D., AND ROSENBLUM, A. H.: Suppurative Intrathoracic Thyroiditis. Arch. Surg. **36:** 867–873, 1938.

30. KROHN, S. E.: Benign Intrathoracic Goiter with Recurring Pleural Effusion. New York State J. Med. **41:** 1767–1769, 1941.

31. LAHEY, F. H.: Intrathoracic Goiter. J. A. M. A. **113:** 1098–1104, 1939.

32. LAHEY, F. H.: Intrathoracic Goiter. S. Clin. North America **16:** 1613–1629, 1936.

33. LAHEY, F. H., AND SWINTON, N. W.: Intrathoracic Goiter. Surg., Gynec. & Obst. **59:** 627–637, 1934.

34. MACLEAN, N. J.: Intra-Thoracic Goiter. Minnesota Med. **11:** 286–291, 1928.

35. MALARD, C.: Étude clinique sur le goitre plongeant ou retrosternal. Thèse, Paris, 1879. Quoted by Clute and Lawrence (8).

36. MASON, J. B.: Mediastinal Goiter (of Aberrant Tissue). Ann. Surg. **116:** 795–798, 1942.

37. MAYO, C. W.: Intrathoracic Goiter. J. Kansas M. Soc. **43:** 405–407, 1942.

38. MEANS, J. H.: The Thyroid and Its Diseases. Philadelphia, J. B. Lippincott Co., 1937, pp. 514–517.

39. NICHOLS, B. H.: Quoted by Crile (11).

40. PAPE, R.: Zur Röntgendiagnose der Struma substernalis. Wein. klin. Wchnschr. **58:** 294–296, 1946.

41. PARSONS, W. B.: Substernal Thyroid. Ann. Surg. **113:** 82–84, 1941.

42. RIVES, J. D.: Mediastinal Aberrant Goiter. Ann. Surg. **126:** 797–810, 1947.

43. SCHLESINGER, M. J., GARGILL, S. L., AND SAXE, I. H.: Studies in Nodular Goiter: Incidence of Thyroid Nodules in Routine Necropsies in Nongoitrous Region. J. A. M. A. **110:** 1638–1641, 1938.

44. SCHULTZE, H.: Über einen Fall von Chylothorax hervorgerufen durch Kompression der Mündungsstelle des Ductus thoracicus durch eine Struma substernalis. Wien. klin. Wchnschr. **38:** 455–456, 1925.

45. SCHWYZER, G.: Diagnosis and Surgical Treatment of Intrathoracic Goitre. J. A. M. A. **74:** 597–601, 1920.

46. SLANY, A.: Zur Kenntnis der echten intrathorakalen Nebenkröpfe. Zentralbl. f. allg. Path. u. path. Anat. **69:** 194–197, 1938.

47. SOLEY, M. H., AND RINEHART, J. F.: Intrathoracic Goiter Simulating Right-Sided Cardiac Enlargement. Am. Heart J. **18:** 237–240, 1939.

48. SWEET, R. H.: Personal communication.

49. WAKELEY, C. P. G.: Goitre Plongeant. Clin. J. **68:** 349–351, 1939.

50. WAKELEY, C. P. G., AND MULVANY, J. H.: Intrathoracic Goiter. Surg., Gynec. & Obst. **70:** 702–710, 1940.

51. WAUGH, J. M.: Relation Between Diseases of Thyroid Gland and Laryngeal Function. In "The Thyroid Gland." Clinics of George W. Crile and Associates. Philadelphia, W. B. Saunders & Co., 2nd ed., 1922, pp. 55–64.

52. VON ZWEIGBERGK, J. L.: Intrathorakale Struma. Acta chir. Scand. **90:** 444–459, 1944.

SUMARIO

Bocio Intratorácico: Su Incidencia, Semiología y Diagnóstico Radiológico

En una serie de 908 tiroidectomías ejecutadas por todas causas, se encontró un bocio parcialmente intratorácico en 20 pacientes y totalmente intratorácico en 8. Veinticuatro de esos 28 bocios eran atóxicos y nodulares, 3 nodulares con leve hiperactividad y 1 reveló carcinoma. No hubo casos de hiperplasia difusa tóxica con exoftalmía. Los enfermos pertenecían al grupo de edad avanzada, teniendo la mayoría mas de cincuenta años. No hubo predominio de sexo.

En el orden de su frecuencia, los síntomas más constantes fueron: edema cervical, disnea de esfuerzo, tos, disfagia, disnea al recostarse, sensación de asfixia y ronquera. Cuatro enfermos se hallaban asintomáticos. En el examen físico, el hallazgo más común e importante consistió en hipertrofia palpable del tiroides, encontrándose en todos los enfermos en los que la difusión intratorácica era parcial. La desviación de la tráquea y la dilatación de las venas del cuello no fueron frecuentes. Un paciente mostraba *goitre plongeant* y otro parálisis de las cuerdas vocales.

Los 28 bocios quedaban en el mediastino superior; 19 eran anterolaterales y 6 posteriores a la tráquea y 3 retroesofágicos.

Los hallazgos roentgenológicos significativos fueron:

(*a*) Desplazamiento de la tráquea por la tumefacción, en 27 casos.

(*b*) Desplazamiento de la tráquea, desde muy alto en el cuello, frecuentemente en la laringe y con alguna inclinación de la laringe.

(*c*) Compresión de la tráquea, frecuente, pero no pronunciada.

(*d*) Desplazamiento o compresión del esófago, asociados a alteraciones semejantes en la tráquea.

(*e*) Movimientos ascendentes del bocio a la deglución, en 84 por ciento de los enfermos estudiados.

(*f*) Calcificación intraestrumosa, en 25 por ciento de los casos.

(*g*) Contorno liso o apenas nodular del tumor.

(*h*) Reflexión de la pleura mediastínica más abajo del bocio.

En todos los casos, se llevó a cabo sin complicaciones la extirpación quirúrgica total, que es el tratamiento de elección para todo bocio intratorácico.

Orthographic Pelvimetry[1]

PAUL C. HODGES, M.D., and RUSSELL L. NICHOLS, M.D.

Chicago, Ill.

BY ORTHOGRAPHIC pelvimetry we mean the production of true dimensional tracings or orthograms of the frontal and lateral views of the pelvis, on which one can measure the transverse diameter of the inlet, the interspinous diameter, the intertuberous diameter, the obstetrical conjugate, the anterior and posterior sagittal of the mid pelvis, the anterior and posterior sagittal of the outlet, and numerous other dimensions, without the aid of computations or specially calibrated rulers. Pelvic orthograms have been used to some extent in the past by van Ebbenhorst Tengbergen (1), Litwer (2), and Thoms (3).

Some years ago, one of us (Hodges), reviewing with Dippel the development of roentgen pelvimetry (4), referred to a 90° triangulation apparatus then about ready for publication and discussed the possibility of developing a linkage pantograph for the making of frontal and lateral orthograms from the pair of 90° roentgenograms that would be produced by that apparatus. During the war the facilities of our laboratory were diverted into other channels, but recently we have completed the apparatus and have devised and constructed the proposed linkage pantograph. With two of our colleagues at the Chicago Lying-In Hospital, we are using this apparatus for pelvic mensuration in 1,000 primiparae and expect eventually to publish our findings relative to the dimensions of the pelvis in eutocia and in dystocia. In the present paper we shall deal merely with the technical details of orthometric pelvimetry.

Our 90° apparatus and pantograph were shown in the scientific exhibit of the Congress on Obstetrics and Gynecology in St. Louis in September 1947, but no account of them has heretofore been published.

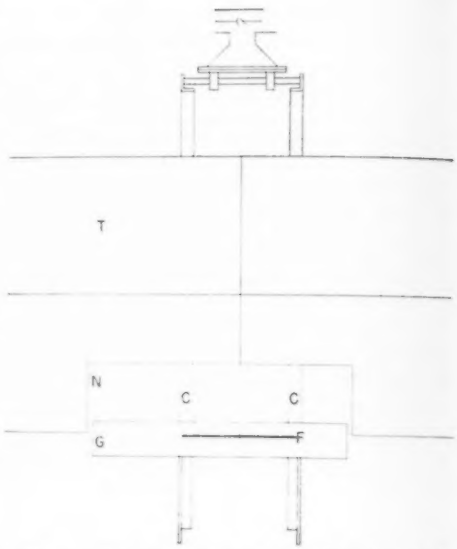


Fig. 1. Schematic plan of central portion of table top (T), notched at the side (N) to receive the grid (G) when the cradle (C) is rotated into position for the making of the lateral film (F).

THE 90° APPARATUS

A motor-driven reciprocating Potter grid and an x-ray tube are attached to a cradle that is mounted beneath the 24 × 84-inch top of a special x-ray table (Fig. 1) which has a 6 × 23 1/2-inch notch cut into its right side to receive the grid during the making of the lateral film. The grid-tube assembly may be rotated so that the tube lies above and the film below for a frontal view (Figs. 2 and 3) or so that the radiation is directed parallel with the floor and the grid is pressed against the side of the patient for the lateral projection (Figs. 4 and 5). In this latter position the grid may be moved inward toward the midline of the table or outward away from that line, the amount of such movement being indicated

[1] From the Division of Roentgenology, The University of Chicago. Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

This work has been aided by a grant from the Illinois Public Health Service.

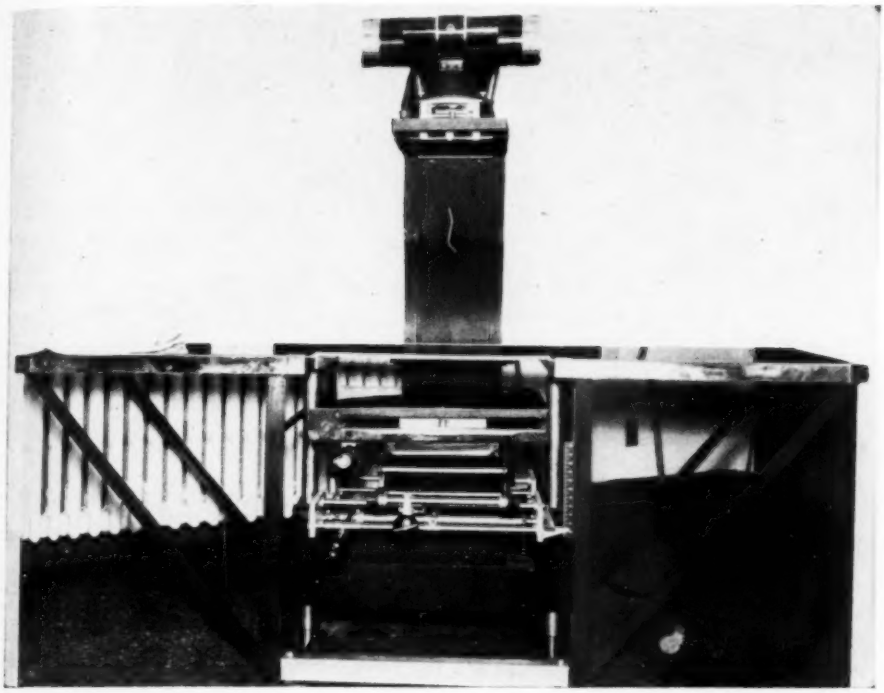


Fig. 2. The 90° apparatus arranged for frontal film. The cradle has been rotated so that tube is above, grid below—preparatory for the making of the frontal film. Before the film is made, the cradle will be moved upward until its top is pressed against the under surface of the table top.

on the H (horizontal) scale. It may also be moved up so that the longitudinal axis of the lateral film moves away from the table top, or downward toward it, the amount of this movement being indicated on the V (vertical) scale.

A stripe down the center of the table locates its midline, and lead wires embedded in the bakelite top of the grid indicate the longitudinal and transverse axes of the films. In both positions, the target of the x-ray tube lies on a perpendicular erected at the center of the film, the target-film distance (D) being 36 inches, and during the making of the frontal film this perpendicular passes also through the midline of the table top.

MAKING THE LATERAL FILM

The patient lies on her back on the table, her right side toward the cut-out in the top, the midline of her body directly over the midline of the table top, with a small pillow between the table top and the lumbar column to reduce the angle θ between the pelvic inlet and the plane of the film, and with the cone of radiation centered about 5 cm. proximal to the upper surface of the pubic symphysis. The degree of angulation of the inlet and the relationship between the center of the cone of radiation and the pubic symphysis do not affect the validity of the measurements, but this is affected by the alignment of the midsagittal plane of the body with the midline of the table. With the patient thus positioned, web straps are crossed over her abdomen and cinched down to hold her in place. A 10 × 12-inch film is now inserted in the grid tray and the cradle is rotated into position for the lateral film. The cradle is pushed toward the midline of the table until the grid presses lightly against the patient's right hip and is raised or lowered until the longitudinal axis of the lateral film lies about halfway between the

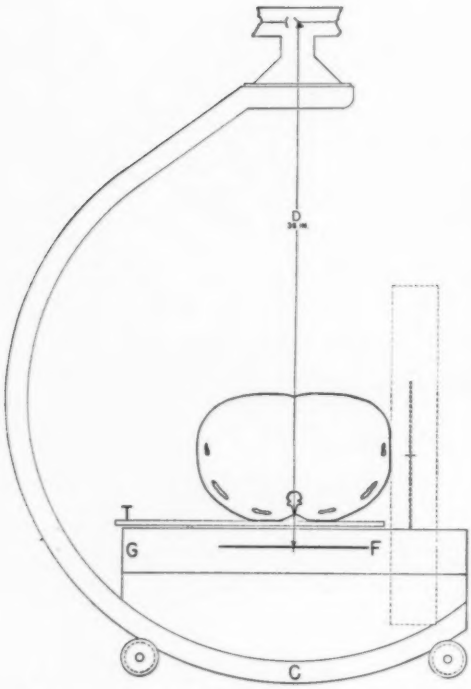


Fig. 3. Geometry of frontal film. Schematic elevation of apparatus in position for frontal film (F). The cradle (C) has been rotated so that the grid (G) lies beneath the table top (T) on which the patient lies.

table top and the upper surface of the pubic symphysis. Horizontal and vertical travel are now locked and the readings on the H and V scales are recorded. The H reading indicates the separation, in centimeters, between the mid-sagittal plane of the body and the plane of the lateral film or, in other words, the *d* of the mid-sagittal plane. The V reading indicates the separation, in centimeters, between the longitudinal axis of the lateral film and the plane that will be occupied by the frontal film. It will be seen presently that the reading on the V scale is employed in determining the *d*'s of the inlet, mid pelvis, and outlet, which are needed for the work-up of the frontal film.

Since the grid is provided with a phototimer, the operator merely initiates the exposure. When the film has received a sufficient amount of radiation, the phototimer automatically terminates it.

MAKING THE FRONTAL FILM

The cradle now is rotated so that the tube lies above and the grid is pressed firmly against the undersurface of the table top. As this is done, a mercury switch automatically cuts out the potentiometer that was calibrated for the lateral film and puts in circuit a second potentiometer that has been calibrated for the frontal film. The exposed film is now removed from the grid tray, a fresh one is inserted, and without more ado the frontal film is made by closing the x-ray switch and holding it closed until the phototimer has terminated the exposure.

THE LINKAGE PANTOGRAPH

An aluminum alloy tracing rod, 3/8 of an inch in diameter and 39 inches long, has a rounded point at its lower end, and a similar writing rod carries a pencil at its lower end. The tracing surface is the flashed opal glass window of a horizontal illuminator, and the writing bed is a bakelite surfaced board which may be raised and tilted relative to the plane of the tracing surface. For plus values of θ the head end is low; for the occasional cases of negative θ the foot end is low. Spring clamps are provided for attaching the film to the tracing bed and the paper to the writing bed (Fig. 6).

Perpendicularly above the center of the tracing surface at a distance of 36 inches (which is the target-film distance of our 90° apparatus) lies the center of a self-aligning bearing. A second bearing of the same type is similarly located relative to the center of the zero position of the writing bed.

A vertical steel supporting post, 3/4 of an inch in diameter and approximately 32 inches long, rising from the center of the wooden top of the instrument, carries a bracket at its upper end on which the upper self-aligning bearings are mounted, provides a support for the central bearing of the parallelogram, and serves also as a guide on which the counterweight for the writing point rides.

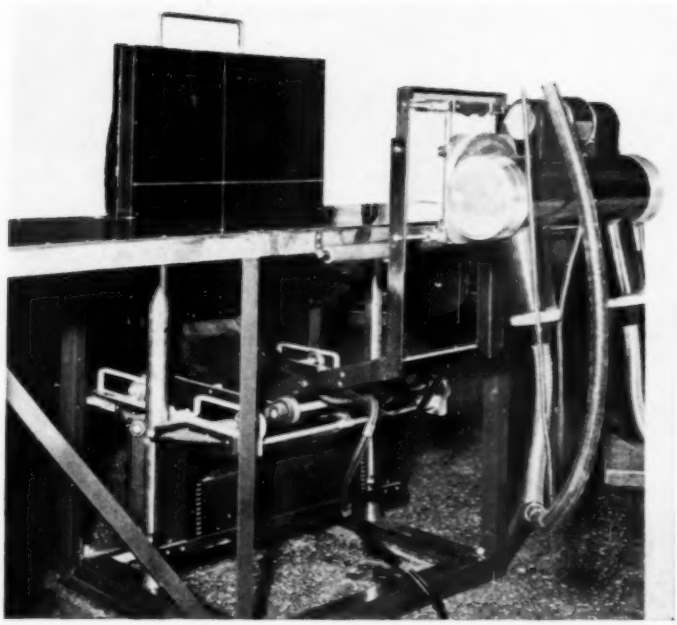


Fig. 4. The 90° apparatus arranged for lateral film. Cradle rotated into position for the making of the lateral film.

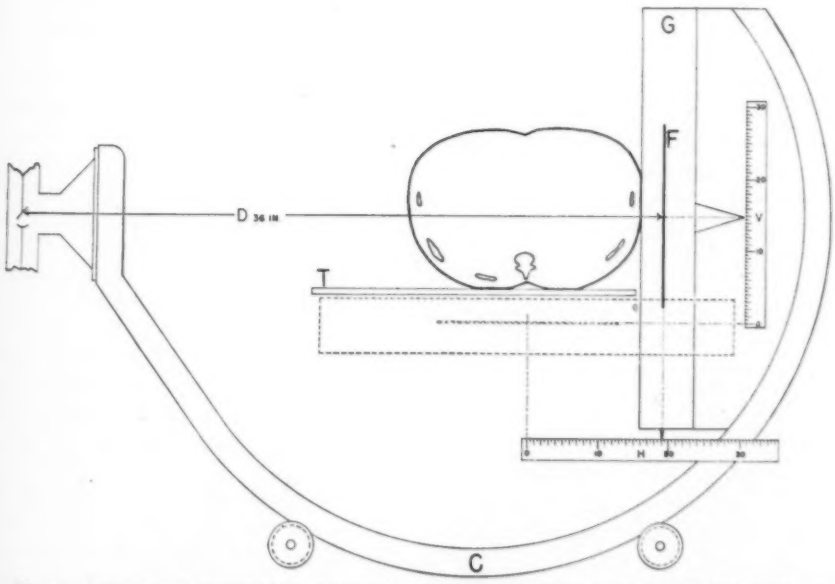


Fig. 5. Geometry of lateral film. Schematic elevation of apparatus with cradle (C) rotated so that the grid (G) is pressed against the side of the patient and the longitudinal axis of the lateral film (F) lies midway between the table top (T) and the anterior surface of the patient's body.

The H scale indicates the distance between the sagittal plane of the body and the plane of the lateral film (the *d* of midline structures in the lateral film). The V scale indicates the distance between the longitudinal axis of the lateral film and the plane that presently will be occupied by the frontal film (shown in dotted lines in the diagram).

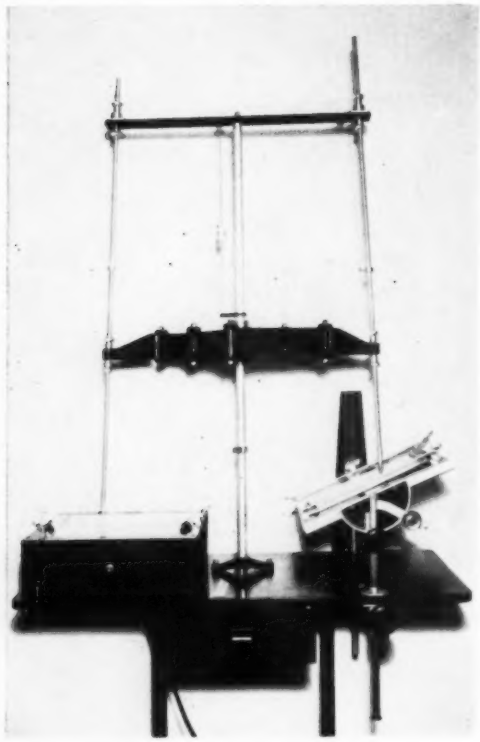


Fig. 6. Linkage pantograph.

If the writing bed (shown at the right) is lowered until its center is at the height of the tracing bed (shown at the left) and rotated until the two beds lie in the same plane, the writing point will duplicate the movements of the tracing point as the latter follows the wax-penciled outlines of structures seen in pelvic roentgenograms.

Raising the writing bed by the object-film distance (d) corrects for divergent distortion and tilting the writing bed to the angle that obtained between the plane of the object and the plane of the film (θ) corrects for oblique distortion.

The perpendicular distance between the surface of the tracing bed and the center of the upper self-aligning bearing through which the tracing rod passes is fixed at the target-film distance (T). The parallelogram (see Fig. 7) forces the writing rod to duplicate the movements of the tracing rod.

THE PARALLELOGRAM

The parallelogram is a rugged, precisely machined 5-membered assembly (Fig. 7) equipped with ball bearings at each of its six articulations. For convenience in description, the parts may be designated as: (1) tracing arm; (2) tracing link; (3) writing arm; (4) writing link; (5) central link. The central link is carried on large ball bearings mounted on a sleeve that is attached by set screws to the supporting post. Usually the parallelogram lies about 14 inches above the tracing surface, but it may be raised or lowered without affecting the geometry of the system. The outer ends of the tracing arm and the writing arm carry self-aligning bearings similar to those that are mounted on the bracket above.

TRACING AND WRITING RODS

The tracing rod passes through the self-aligning bearing in the tracing link of the parallelogram and the corresponding upper fixed bearing, and its point is held a few centimeters away from the tracing surface by a light coil spring. The operator presses the point down lightly against the surface of the film while the tracing is being done, and the spring raises the point away from the film when the tracing has been completed.

The writing rod passes through the self-aligning bearing in the writing arm of the parallelogram and the corresponding upper bearing and is counterbalanced by a light nylon fishing line attached to a collar on the rod and passing over pulleys to be anchored to a counterweight riding on the supporting post. A second nylon line, hanging down from the counterweight, is used by the operator to lift the writing point away from the bed or lower it onto the writing surface. A friction catch anchors this second line to hold the writing point away from the bed when the instrument is not in use.

GEOMETRY OF THE PANTOGRAPH

The parallelogram itself has a 1:1 linkage, so that movements of the tracing arm are exactly duplicated by the writing arm except that the latter are upside down. Reduction in size of the tracing to correct for divergent distortion in the film is accomplished by raising the writing bed, thus reducing the length of the portion of the writing rod that protrudes through the bearing in the writing arm of the parallelogram; oblique distortion is corrected by tilting the writing bed until its angle rela-

tive to the tracing bed is the same as the angle between the object and the film.

As the operator carries the tracing point over the outline of some particular image in a roentgenogram, the tracing rod defines in space the cone of radiation that must have produced that image. If that cone could be cut through by a plane located at the height (d) and the angle (θ) occupied by the object at the time the sacrum and pubic symphysis, the mean position of the iliopectineal lines, the mean position of the ischial spines, and the mean position of the ischial tuberosities are drawn onto the lateral pelvic film, which is then attached to the tracing bed of the pantograph with "head," "foot," "right," and "left" corresponding with the labels on the instrument and the long and short axes of the film coinciding with the

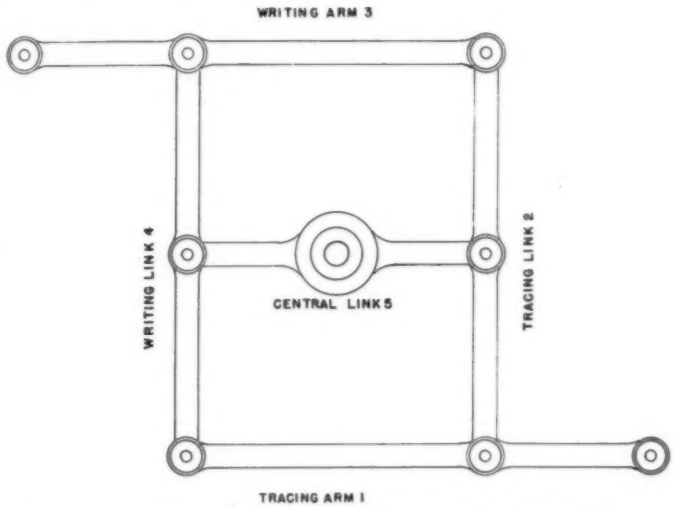


Fig. 7. The parallelogram of the pantograph. Ball bearings allow extremely free movement of the central link (5) about the central supporting post, at the junctions between the central (5), the tracing (2) and the writing (4) links and at the four corners of the rectangle. The parts are made light and yet rigid to minimize sagging and maintain the plane of the parallelogram parallel with the plane of the tracing bed. The bearings on the outer ends of the tracing arm (1) and the writing arm (3) are self-aligning to allow free angulation of the tracing and writing rods.

raying was done, the circumference of that section of the cone would correspond exactly with the circumference of the object. Linking the tracing rod and the writing rod together by means of the parallelogram forces the latter to duplicate exactly the movements of the former. Raising the writing bed to the d of the object and tilting it to the θ of the object define the plane of the desired section of the cone of radiation and the writing point describes on that plane the circumference of the desired section.

PRODUCTION OF THE LATERAL ORTHOGRAM

With a wax pencil the outlines of the cross lines on the opal glass. The "mean position" of the non-midline structures is a reasonable approximation of the positions that their shadows would take on the film if the structures in question were located in the mid-sagittal plane of the patient's body. For example, there are two shadows of the sciatic notch, ischial spine, and ischial tuberosity—one cast by the right side of the pelvis, which is close to the film; the other by the left side of the pelvis, which is relatively far from the film. Both shadows are sketched lightly on the film with a wax pencil and then a third empirical line is drawn midway between the other two, after which the original two are erased.

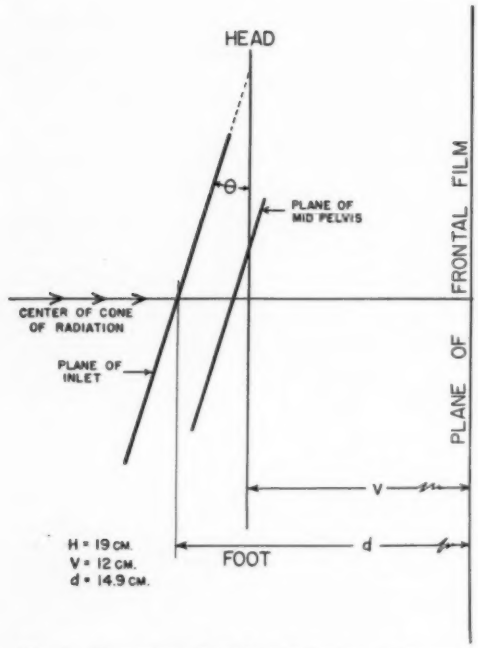


Fig. 8. Geometry of pelvic orthograms. Schematic representation of a lateral orthogram in which the plane of the pelvic inlet and the plane of the mid pelvis are indicated by heavy lines. The tracing shows these structures in true size and in true relationship to other parts because it was made with the tracing bed set with θ zero and d at 19 cm., which was the reading on the H scale at the time the film was made.

The θ of the inlet is measured with a protractor and the d's of inlet and mid pelvis are obtained by measuring along the axis of radiation from the center of the film to the location of the mid pelvis and the inlet and then adding to these measurements the recorded value of V.

This empirical line includes the mean position of the ischial spines and ischial tuberosities.

After a piece of ordinary typewriter paper has been attached to it, the writing bed is raised above its zero position by the number of centimeters recorded as the H scale reading of the 90° apparatus and tilted until its protractor reads zero, showing that it is parallel with the tracing bed. When the tracing point has been placed on some selected point on the wax-pencil outline (for example, the sacral promontory), the writing point is lowered onto the paper and the outline of the sacrum is sketched in. Raising the writing point as he moves the tracing rod to each new starting point, the operator continues until he has developed on the paper a true dimensional outline or orthogram of all the structures outlined in wax on the film.

WORK-UP OF THE LATERAL ORTHOGRAM

Figure 8 is a schematic diagram of a lateral orthogram made from a theoretical lateral roentgenogram in which the planes of the pelvic inlet and of the mid pelvis are represented by heavy oblique lines. The anteroposterior diameters of the inlet and mid pelvis and the θ of the inlet are measured directly by means of a ruler and a protractor. Measurement of the d's of the inlet and mid pelvis, which are needed for the making of the frontal orthogram, also may be obtained without computations. Since the V recorded at the time the film was made was 12 cm., then by definition the plane of the frontal film lies 12 cm. behind the longitudinal axis of the lateral. To obtain the d of the inlet, therefore, one places a ruler along the transverse axis of the lateral orthogram with its 12-cm. point at the intersection of the longitudinal and transverse axes and at the point where the inlet crosses the ruler reads the d of the inlet—in this case 14.9 cm. In the same manner the d of the mid pelvis is read off.

Figure 9 is an actual lateral orthogram on which are indicated: (1) the obstetrical conjugate; (2) anterior sagittal of the mid pelvis; (3) posterior sagittal of the mid pelvis; (4) anterior sagittal of the outlet; (5) posterior sagittal of the outlet.

The broken diagonal line drawn from the posterior surface of the pubis through the mean position of the iliopectineal line to the anterior surface of the sacrum is the plane of the pelvic inlet. When this line has been established, a parallel ruler is placed with one bar on the line and the other coinciding with the mean position of the ischial spines. A second broken diagonal line is then drawn parallel with the first and finally, the second limb of the parallel ruler having been moved to the mean position of the ischial tuberosities, a third broken diagonal line is drawn. Since in this case the recorded V is 16 cm., a ruler is placed along the transverse axis with its

16-cm. mark at the center of the film and *di* (the object-film distance of the inlet), *dm* (object-film distance of the mid pelvis) and *do* (object-film distance of the outlet) are read off directly.

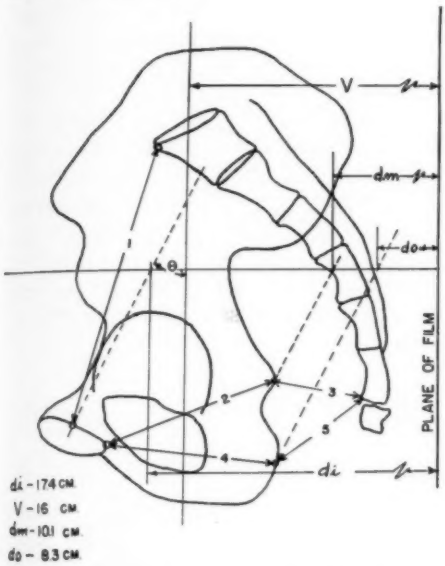


Fig. 9. Lateral orthogram. The lateral orthogram serves two purposes:

(*A*) It allows direct measurement of such pelvic dimensions as

(1) The obstetrical conjugate
(2) Anterior sagittal of mid pelvis
(3) Posterior sagittal of mid pelvis
(4) Anterior sagittal of outlet
(5) Posterior sagittal of outlet

(*B*) It supplies the following values needed for working up the frontal film:

θ = the inclination of the plane of the inlet relative to the plane of the frontal film
di = the height of the inlet above the frontal film
dm = the height of the mid pelvis above the frontal film (measured at the ischial spines)
do = the height of the outlet above the plane of the frontal film (measured at the ischial tuberosities)

MAKING THE FRONTAL ORTHOGRAM

The pelvic inlet, ischial spines, ischial tuberosities, and the two axes are outlined in wax pencil on the frontal film, which is then attached to the tracing bed. After paper has been attached to it, the writing bed is raised until its vertical scale stands at the *d* of the inlet (*di*) and tilted head end low, foot end high, until the protractor reads the θ of the inlet.

After the tracing point has been placed on the pubic symphysis and the pencil has been lowered onto the paper, the operator traces along one side of the inlet as far as the sacral promontory and then, raising the pencil as he returns the point to the symphysis, traces the other side of the inlet. With this maneuver the pencil is always moving "down hill" as it writes, so that lateral pressure on the rod bearings is reduced, less spring occurs in the rods themselves, and less error is introduced by lost motion in the system.

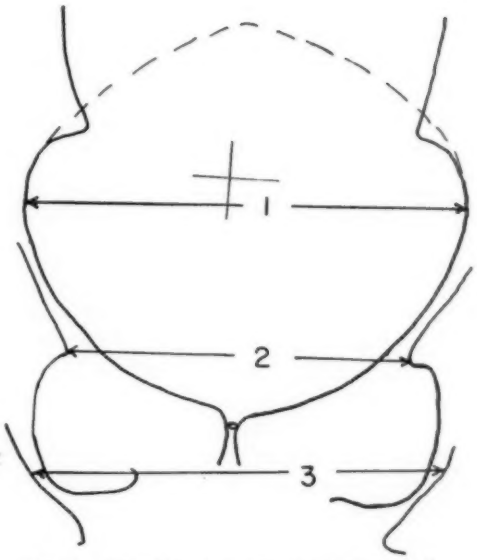


Fig. 10. Frontal orthogram. 1. Transverse diameter of inlet. 2. Interspinous diameter of mid pelvis. 3. Intertuberous diameter of outlet.

After the tracing of the inlet is completed, the writing bed is lowered to the *d* of the ischial spines and then to the *d* of the ischial tuberosities for the tracing of the spines and tuberosities. For both of these tracings the bed remains tilted to the θ of the inlet.

WORK-UP OF THE FRONTAL ORTHOGRAM

The transverse diameter of the inlet, interspinous diameter, and intertuberous diameter are now drawn and measured (Fig. 10).

PROBABLE DEGREE OF ACCURACY

As is true of all forms of x-ray pelvim-

etry, the accuracy of this method can be no better than the accuracy with which the parts can be identified in the roentgenograms. Since the patient lies flat on her back throughout the examination, full lateral and full frontal positions are assured and in the lateral roentgenogram the pubic symphysis, the iliopectineal lines, and the entire sacrum usually are well seen. The ischial spines normally show well in this view unless they are unusually thin or blunt, and even then by following the curves of their bases one can sometimes make a reasonable guess. The main difficulty in the lateral film is the location of the ischial tuberosities.

In the frontal film the end-points of the intertuberous diameter usually can be identified with considerable assurance as the centers of dense crescentic lines that show through the shadows of the other portions of the tuberosities. It is almost never possible, however, to see these lines in the lateral film. Furthermore, the direct inspection of numerous dried pelves leads Dippel to conclude that there is no regularly dependable relationship between them and the rounded inferior margins of the tuberosities, which show so well in the lateral view. Though we recognize the shortcomings of the procedure, we have been forced to use the mean position of the centers of these curved lower surfaces as the end-points of the anterior and posterior sagittal diameters of the outlet. In spite of this unavoidable empiricism, our measurements on dried pelves are gratifyingly precise and evidence is accumulating to indicate that they are reasonably dependable also in the living subject.

Radiologists who do pelvimetry only infrequently probably will consider the 90° apparatus and pantograph too costly, but where the volume of work is considerable we believe that frontal and lateral orthograms will prove to be not only rapid, convenient, and for the most part accurate, but also economical because of the saving in time.

Division of Roentgenology
University of Chicago
Chicago 37, Ill.

REFERENCES

1. VAN EBBENHORST TENGBERGEN, J.: In Supplement III to Acta radiologica, 1928, p. 57.
2. LITWER, H.: Roentgen Pelvimetry. J. Obst. & Gynaec. Brit. Emp. **43:** 1158–1161, 1936.
3. THOMS, H.: Pelviscope: Reducing Apparatus for Grid Method of Pelvimetry. Radiog. & Clin. Photog. **13:** 10–11, 1937.
4. HODGES, P. C., AND DIPPEL, A. L.: Use of X-Rays in Obstetrical Diagnosis, with Particular Reference to Pelvimetry and Fetometry. Internat. Abstr. Surg. **70:** 421–446, 1940; in Surg., Gynec. & Obst., May 1940.

SUMARIO

Pelvimetría Ortográfica

Con el aparato de triangulación de 90° y el pantógrafo de enlace, aquí descritos, pueden determinarse los siguientes diámetros pelvianos sin computaciones de ningún género: diámetro transversal del estrecho superior, diámetro interespinoso de la pelvis media, diámetro intertuberoso del estrecho inferior, diámetro anteroposterior del estrecho superior, sagital posterior de la pelvis media y sagital posterior del estrecho inferior.

Con este procedimiento se estudiaron las dimensiones pelvianas en 1,000 primíparas. Si bien los resultados no eran completos para la fecha de la preparación de este trabajo, los datos ya obtenidos permiten recomendar el método como rápido, conveniente, económico y, en conjunto, exacto.

DISCUSSION

Paul C. Swenson, M.D. (Philadelphia, Penna.): I want briefly to commend Drs. Hodges and Nichols on a very clever and ingenious new method of arriving at the measurements of the pelvis. We all know that anything that comes out of the laboratory of Dr. Hodges is of the highest degree of precision.

I have no comments to make about the technic with one exception. I would like to ask Dr. Hodges whether or not he is considering trying this procedure in the erect position. Since Drs. Golden and Ball recently popularized the erect position, I have found it essential for a good appraisal of the head size *versus* the pelvic inlet, as well as other factors pertaining to the course of labor. It seems to me that this is a most important point. The value of this new method of Dr. Hodges and Dr. Nichols will be apparent with its eventual application to the thousand cases that they intend to study.

Dr. Hodges (*closing*): Dr. Swenson has raised the question of the advisability of using the erect position in making the lateral pelvic roentgenogram. We did use that procedure for a good many years, and I am acquainted with its advantages. To my mind, however, one great disadvantage of the erect position outweighs all of its advantages. I refer to the fact that in the erect position gravity causes the abdominal tissues to sag down and thereby increases the amount of tissue that has to be penetrated. In the horizontal position, however, gravity plus a compression band can be used to force flabby tissues out of the way, thereby decreasing the thickness of the part.

We have now examined approximately 800 out of the 1,000 that will make up our first series and presently will publish the results. The purpose of this paper was merely to put on record the method that we are employing.

Hydatid Disease[1]

S. F. OOSTHUIZEN, M.D., F.R.C.P., D.M.R., and M. H. FAINSINGER, M.B., B.Ch., D.M.R.D.

Department of Radiology, University of Pretoria
Pretoria, South Africa

HYDATID DISEASE (echinococcosis) does not present a constant clinical pattern, and consequently the clinical diagnosis tends to be inaccurate. As is so often the case, failure to think of the condition rather than lack of knowledge about it accounts for most of the misdiagnoses. It would appear, therefore, of some value to re-emphasize certain of the features of hydatid disease, more particularly from the radiological point of view.

In South Africa, as in Australia and New Zealand, Argentine, Uruguay, Iceland, and the Middle East, hydatid disease is very common. Its distribution is directly related to its hosts, members of the dog family, which harbor the worm, and sheep, cattle, man, and other animals in which the hydatid phase of the parasite's life cycle is passed.

Much of the original work on hydatid disease was done in France by François Dévé, who in 1913 published a review of 2,727 cases.

Barnett (1943), of New Zealand, studied 1,617 cases occurring in Australia and New Zealand and drew attention to a higher incidence of pulmonary hydatid disease (23 per cent) than found by Dévé (8.5 per cent). In Iceland, on the other hand, involvement of the lungs according to Claessen is remarkably infrequent.

Although no series of cases of similar size has yet been accumulated in South Africa, our impression is that pulmonary hydatids are common, and the incidence here probably does not differ significantly from that stated by Barnett for Australia and New Zealand.

The cestode *Echinococcus granulosus* (*Taenia echinococcus*) is a parasite of the dog family and in South Africa is widespread among farm dogs, wild dogs, and jackals. These animals become infested by eating raw offal, particularly lungs and liver, derived from sheep, cattle, or equines which may harbor hydatid cysts. As far as domestic dogs are concerned, prevention of infestation is simple, consisting merely in not feeding them such offal in the raw state. In fact, domestic dogs are not a frequent source of hydatid disease in urban areas.

Eggs from the cestode are ingested by man after contamination of the hands by handling dogs. Dévé describes echinococcosis as "a disease of dirty hands." Children are the usual victims, probably because their hands are more frequently in their mouths. Dew states that in most cases a hydatid cyst is nearly as old as the patient.

The embryo reaches the liver by means of the portal blood stream after penetrating the mucosa of the upper intestinal tract. About 70 per cent of hydatids lodge in the liver and develop there. Those that pass the liver are likely to travel via the right side of the heart to the lungs, which are second to the liver in frequency of involvement. Finally, a few embryos pass through the lungs and lodge in the systemic distribution, as in brain, bones, or kidneys.

The embryo develops into the hydatid cyst, the structure of which is well known. The tissues of the host react to form an ectocyst, which is not attached to the embryo's endocyst, and from which the latter is readily stripped surgically, or on rupture in other ways.

Arias Bellini has drawn attention to certain differences in the development of the hydatid in bone. When developing in a restricted space, as in the interstices of bone trabeculae, the hydatid does not

[1] Accepted for publication in July 1948.

form the usual cyst, but undergoes proliferation of the germinal layer, with the formation of microvesicles. The hydatid tissue shapes itself according to the crevices in which it grows, gradually eroding the bone by pressure atrophy. A cystic appearance is produced only late in the disease, when there has been extensive bone destruction. This process of exogenous vesiculation is the characteristic feature of hydatid disease of bone, and accounts for the lack of similarity of its appearances and that of the disease elsewhere. Bone hydatids are relatively rare, occurring in about 1 per cent of cases of hydatid disease (Arias Bellini).

Hydatids are frequently multiple. Dew estimates that 60 per cent of *primary* hydatids are multiple, but other authorities do not place the figure as high as this. Multiple *secondary* hydatids may develop in the pleural or peritoneal cavities or elsewhere, following leakage or rupture of an adjacent cyst. They also follow contamination at operation or on paracentesis. Metastatic hydatids may follow rupture of a cyst into a blood vessel, but this must be a pathological curiosity. A single case of implantation hydatid in soft tissues following a dog bite is recorded by Toole.

Inhalation of ova in dust-laden air is probably a frequent mode of propagation in arid areas devoted to sheep farming. In the dry Karroo area of South Africa, this method of spread probably accounts for the greater frequency of hydatid disease in this area than in less arid parts of the country. A similar theory was advanced by Bird in 1877 to account for the frequency of hydatids in such areas in Australia.

CLINICAL FEATURES

There is no constant clinical picture of hydatid disease. The symptoms and signs depend largely on the location of the cyst.

In the liver, the majority of cysts are silent, but they may leak into a bile duct, discharging their contents and leading to an obstructive jaundice with biliary colic, which is usually mistaken for calculous cholecystitis. They may leak, also, into the pleural cavity or even into the bowel, when a spontaneous cure may be effected. A palpable, rounded smooth swelling may be found and lead to a correct diagnosis, but this is not common. Even rarer is hydatid fremitus, found on palpation and ascribed to the jarring of the daughter cysts against the endocyst. This is said by Barnett (1939) to occur in less than 1 per cent of cases. In order to give any signs, the cyst must be superficial.

Cysts in the lung are also frequently silent. They may cause pressure effects, atelectasis and bronchiectasis, and are prone to infection, with the development of what is virtually an abscess. The cyst may involve the pleura and be concealed by the resultant effusion, and daughter cysts may develop here. Hydatid disease is not infrequently associated with chronic cough and otherwise unexplained hemoptysis (Fig. 5). Occasionally the cyst ruptures into a bronchus, with spontaneous resolution.

When a bone is involved, the diagnosis is not easily made clinically. A vague clinical picture, with "rheumatic" pains, may continue for years, until the hydatid erupts through the cortex, with the development of a soft-tissue mass and finally of a sinus and secondary osteomyelitis.

Involvement of the brain frequently allows of early localization neurologically, although it does not necessarily permit of identification of the nature of the mass. Demonstration of hydatids elsewhere may offer presumptive evidence.

Rupture or leakage of cysts into tissue spaces or body cavities may be associated with severe anaphylactic reactions, which may prove fatal. Gradual seepage may cause prolonged toxic manifestations, which are likely to mislead the investigating clinician.

LABORATORY FINDINGS

Two laboratory tests are of value in confirming the diagnosis.

The Casoni skin sensitivity test, with treated cyst fluid as the antigen, is widely

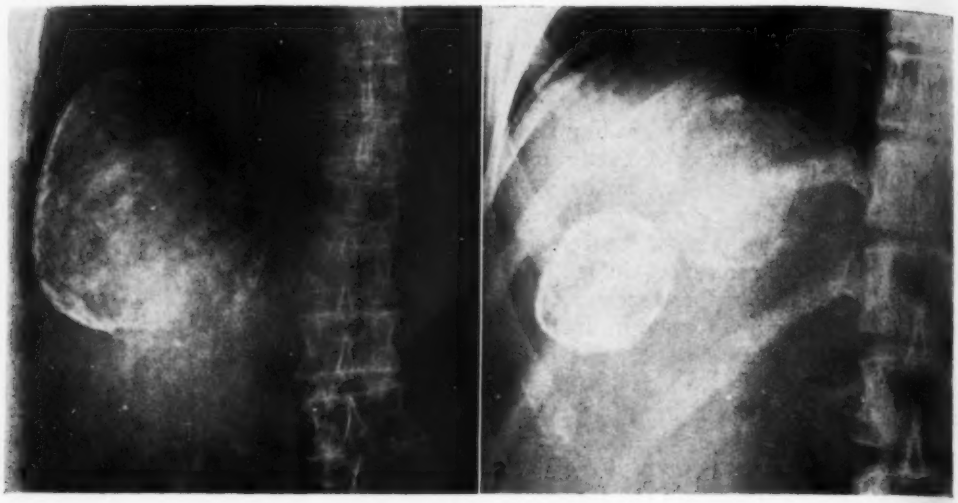


Fig. 1. Large calcified hydatid cyst of the liver.
Fig. 2. Smaller cysts in the liver, showing progressive calcification.

used and is positive in over 90 per cent of cases when the antigen is well prepared and reliable. False positives may occur, but it is difficult to rule out a small, possibly dead, cyst in such cases. A false negative result may be due to faulty antigen or to an early infestation with poor antibody response.

The complement-fixation test (Weinberg) is slightly more involved, but in doubtful cases is of value as a supplement to the skin test.

Eosinophilia is present in active hydatid disease, and a differential white cell count over 5 per cent is usually found.

RADIOLOGIC STUDIES

A hydatid cyst becomes manifest by virtue of its mural calcification, by its own radiopacity, or by displacement or destruction of adjacent structures. The diagnosis is not always possible with certainty, and frequently one cannot go further than to indicate the presence of a mass. A calcifying, roughly spherical tumor is always highly suggestive of hydatid cyst, and the presence of concentric or polycyclic calcifications due to calcium deposition in the walls of the daughter cysts is, for practical purposes, pathognomonic.

The following comments on hydatid cyst in particular situations are, to a large extent, illustrated by the accompanying figures, which are from proved cases of hydatid disease.

Liver: Being the commonest site, the liver should be examined radiologically in all suspected cases of hydatid disease. The following evidence may be elicited: (1) calcification (Figs. 1 and 2); (2) enlargement of the liver shadow; (3) elevation of the right dome of the diaphragm, with decreased movement; (4) disk atelectasis at the right lung base. In cases where calcification has occurred, the presence of an opaque ring shadow supports the diagnosis, but endocyst calcification must be distinguished from calcification in abscesses of the liver, in tuberculomata, neoplasms, blood vessels, gallstones and gallbladder and from calcified lesions of the upper pole of the right kidney, of the suprarenal, and of the head of the pancreas.

In the cases where no calcification is present, only the last three of the signs listed above may be elicited. In this event, no absolute diagnosis is possible, as similar appearances may be produced by any other mass in the liver.

Lung: In the case of the lung, the presence of a tumor is far more readily

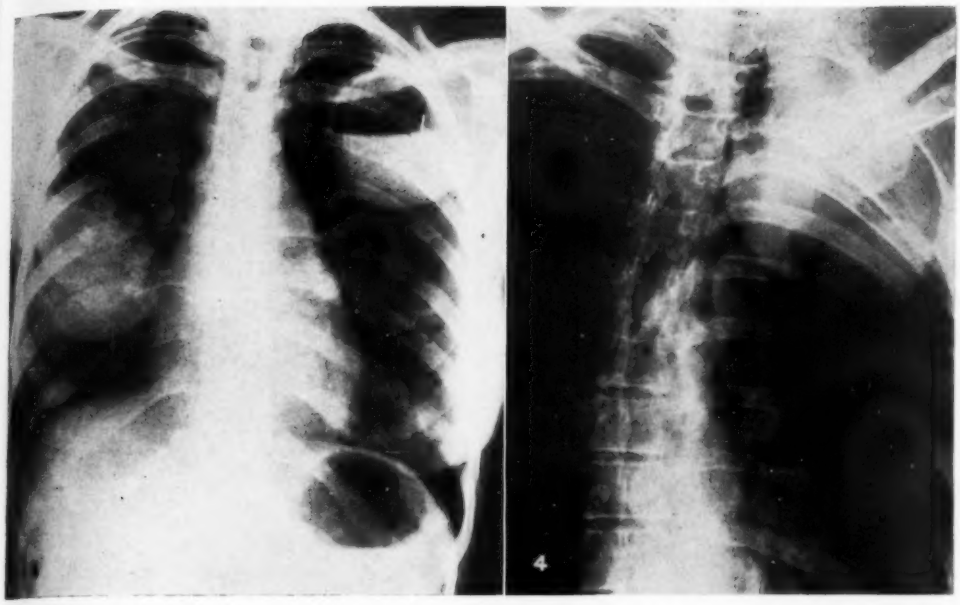


Fig. 3. Two pulmonary hydatids, the one in the right lung presenting as a rounded opacity, while that in the left shows a partial ring shadow due to its communication with a bronchus.
Fig. 4. Apical pleural hydatid, causing pressure erosion of second left rib, simulating a malignant neoplasm.

apparent, but identification of its nature may be very difficult. Hydatids of the lung may reasonably be divided into five groups according to their radiological appearances:

(1) *The Rounded Opacity* (Fig. 3): In the absence of calcification, a rounded mass due to a hydatid may be mistaken for numerous other lesions, particularly benign and malignant neoplasms, and metastatic deposits. Elongation of the cyst on inspiration and a return to the rounded shape on expiration (Escudero-Nemenow sign) may give a useful clue as to the fluid nature of the lesion, although the sign is not specific for hydatid cysts.

(2) *The Ring Shadow* (Fig. 3): The hydatid may rupture into a bronchus, when infection usually supervenes. The picture at this stage is that of a ring shadow. The ectocyst shadow is well defined and presents a regular outline of constant thickness throughout its circumference. The collapsed endocyst may be apparent lying within the pulmonary ectocyst cavity. Healing may follow expectoration of the cyst wall, but this favorable outcome is not frequent. The endocyst may be seen floating on top of the free fluid in the cyst, and showing the sign of the camalote (Jenkins) (Fig. 5).

The differential diagnosis at this stage must be made from lung abscess, tuberculous and other cavities, congenital cysts, and interlobar effusions and empyemata. The associated features of the latter group usually allow of their differentiation.

(3) *Calcified Opacities:* In the presence of calcification, the diagnosis may be easier, although calcification in dermoid cysts, in old abscesses, in tuberculous foci, and occasionally in neoplasms, may cause very similar appearances.

(4) *Opacities Abutting on the Pleura* (Fig. 4): A hydatid cyst arising at the pleural surface is likely to give rise to a hemispherical opacity. This type must be distinguished from an encysted pleural effusion, from a pleural neoplasm or metastasis. There may be involvement of bone, or erosion by pressure.

(5) *Opacities Abutting on the Mediasti-*

Fig. 5. Tomogram of a hydatid cavity communicating with a bronchus. The collapsed endocyst, lying within the ectocyst cavity, and the irregular draining bronchus from which hemorrhage occurred are shown.

num: When the cyst develops in close proximity to the mediastinum, it can closely simulate a dermoid cyst, enlargement of mediastinal lymph nodes or other mediastinal structures.

In the Abdomen, Elsewhere Than in the Liver: Hydatids in the abdomen elsewhere than in the liver are not frequent. They must be differentiated from visceral calcification due to other causes, particularly in cyst walls. Thus, in the spleen, differentiation from splenic cysts of congenital or traumatic origin may prove difficult.

In the kidney, hydatids may be recognized in the pyelogram by distortion by a spherical mass (Fig. 6), although a simple solitary cyst, or even a malignant neoplasm, may produce a similar deformity. In the presence of mural calcification, the distinction becomes easier.

A spherical mass in the abdomen may be apparent from displacement of the bowel on barium meal examination. A hydatid of the head of the pancreas may thus be detected by a widening of the duodenal loop (Fig. 7). This must be distinguished from other cysts and from tumors in this position. In the normal hypersthenic transverse stomach, the loop also appears wide, but the absence of narrowing of the lumen, and the absence of obliteration of the plicae circulares will differentiate this normal condition.

Secondary implantation hydatids of the peritoneum are not easy to recognize radiologically but may be identified by pneumoperitoneum. Differentiation from peritoneal metastases is difficult. These

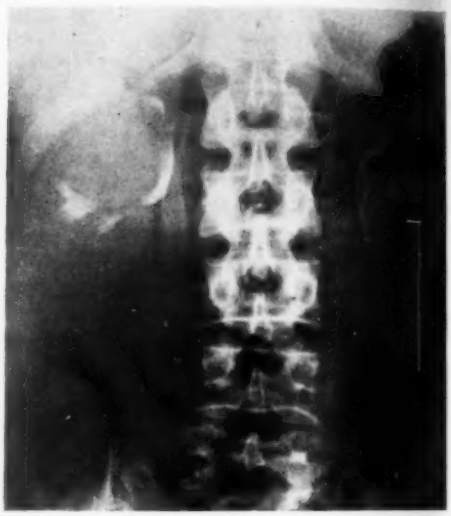


Fig. 6. Intravenous urogram, showing a large rounded filling defect due to a renal hydatid.

cases are not usually seen with calcification, due to rapid deterioration in this stage.

Central Nervous System: Radiologically, a cerebral hydatid may present as a calcified plaque or node in any part of the cranial cavity. The more extensive the calcification, the easier the recognition. With air studies, displacement of the ventricles by a spherical mass is likely to be demonstrated. This mass must be differentiated from other intracranial tumors.

The pattern of the calcification is sometimes of value in identification; a crenated ring shadow is frequently found in a partially collapsed cyst and may be rather characteristic (Figs. 8 and 9). In the majority of cases, however, with or without calcification, a differential diagnosis from other intracranial masses is not possible.

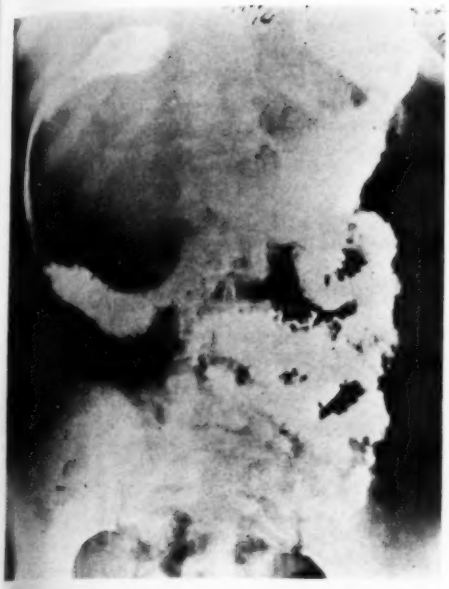


Fig. 7. Film obtained after a barium meal, showing marked widening of the duodenal loop and narrowing of the duodenal lumen, due to a hydatid of the head of the pancreas.

In the spinal cord, hydatids may occur in the meninges and produce widening of the interpedicular space in the antero-posterior view (Fig. 10) and hollowing of the posterior surface of the vertebral body in the lateral view. These findings are not distinguishable from those similarly produced by other expanding lesions in a like situation. Myelography does not usually elucidate the etiology of the mass causing the obstruction.

Bones: In long bones, the lesion usually starts as an erosive condition of the medulla, when it must be distinguished from pyogenic osteitis, Ewing's tumor, and metastasis (Fig. 11). As it develops, it may form well defined cavities which expand the bone and which have to be differentiated from other causes of transradiant expansion, including simple bone cyst, giant-cell tumor, fibrous dysplasia, hyperparathyroid cyst, eosinophilic granuloma, and rarely a bone abscess. The hydatid tends to break through the cortex into the soft tissues, and the erosion, with the soft-tissue mass, may produce a picture much like that of a primary sarcoma of bone. Secondary infection is frequent and, once it supervenes, detection of the primary lesion underlying the secondary osteitis is not always possible.

Other Tissues: Echinococcosis in other tissues, such as muscle, may give no radiological evidence other than soft-tissue swelling. If calcification be present, diagnosis may be easy. Cysts in this position tend to be molded by the lines of pressure,

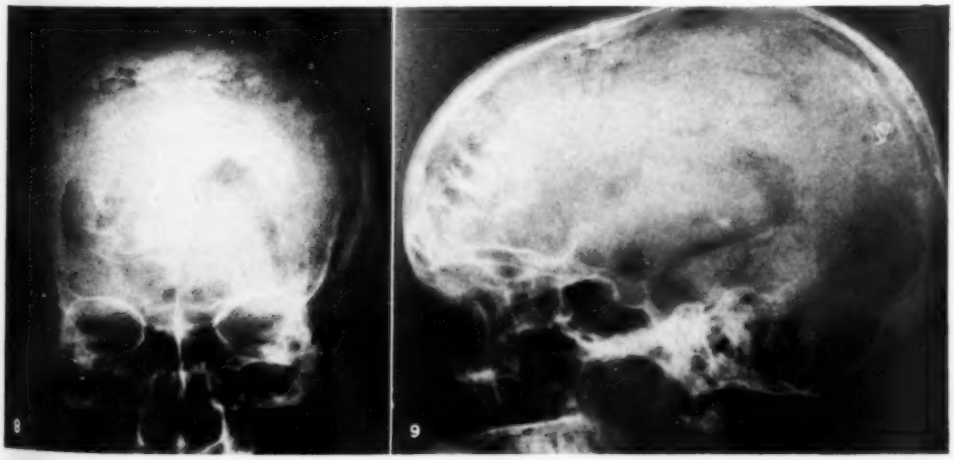


Fig. 8. Calcified hydatid of the frontal lobe of the brain, with crenation of its wall. The right ventricle, which is filled with air, shows pressure deformity.
Fig. 9. Lateral projection of the case shown in Fig. 8.

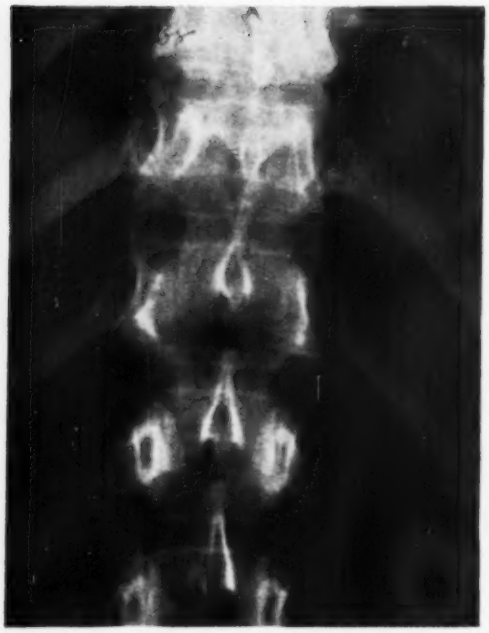


Fig. 10. Pressure erosion of both pedicles of the twelfth thoracic vertebra, with resultant widening of the interpedicular spaces, due to a hydatid cyst of the spinal meninges.

and they may become elongated and stretched. Occasionally, they produce pressure effects on adjacent bone.

SUMMARY

1. A short review of the literature with reference to the etiology, pathology, and clinical features of hydatid disease in general and as it involves particular organs and tissues is given.

2. The radiologic findings based on the appearances in a number of proved cases are described and the differential diagnosis is discussed. Difficulties in differentiation from other conditions are stressed.

REFERENCES

ARIAS BELLINI, M.: Osteohydatidosis: Its Radiological Features. Radiology **47:** 569–574, December 1946.

BARNETT, L.: Hydatid Cysts: Their Location in the Various Organs and Tissues of the Body. Australian and New Zealand J. Surg. **12:** 240–248, April 1943.

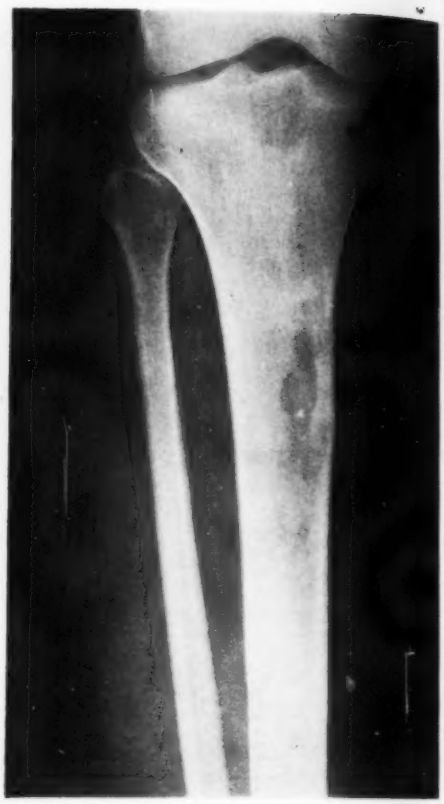


Fig. 11. Hydatid disease of bone, with erosion of the cortex from within and extension of the process down the medulla.

BARNETT, L.: Hydatid Disease: Errors in Teaching and Practice. Brit. M. J. **2:** 593–599, Sept. 16, 1939.

BIRD, D.: Hydatids of the Lung. 1877. Quoted by Barnett, 1943.

CLAESSEN, G.: On Echinococcosis in Lung. Acta radiol. **16:** 601–615, 1935.

DÉVÉ, F.: Les localisations de l'echinococcose primitive chez l'homme. Compt. rend. Soc. de biol. **74:** 735, 1913.

DEW, H. R.: Hydatid Disease; Its Pathology, Diagnosis and Treatment. Sydney, Australia, Australasian Medical Publishing Co., 1928.

JENKINS, J. A.: Pulmonary Hydatid Disease: Sign of the Camalote. Australian & New Zealand J. Surg. **15:** 296–298, April 1946.

TOOLE, H.: Echinococcus der Glutealmuskeln nach Hundebiss am Gesäss. Arch. f. klin. Chir. **184:** 183–184, 1935.

Pretoria Hospital
Pretoria, South Africa

SUMARIO

Hidatidosis

Este breve repaso de la literatura comprende etiología, anatomía patológica y características clínicas de la hidatidosis en general y en sus manifestaciones en ciertos órganos y tejidos.

Al describir y reproducir los hallazgos radiológicos basados en el aspecto de varios casos comprobados, se discute el diagnóstico diferencial. Un tumor calcificado toscamente esférico es siempre muy indicativo de quiste hidático, y la presencia de calcificaciones concéntricas o policíclicas resulta, para fines prácticos, patognomónica.

Spontaneous Hemopneumothorax

Etiological Considerations and Case Report[1]

JULIUS SOLOVAY, M.D.

Veterans Administration Hospital, Montgomery, Ala.

BLOOD OR BLOODY fluid, with or without air, commonly occurs in the pleural cavity as a result of several different pathological conditions, chief among which are traumatic laceration of intercostal or pulmonary vessels, tuberculosis, and cancer. Unusual causes are rupture of a thoracic aneurysm and infarction of the lung with aseptic softening, perforation and hemorrhage into the pleural cavity (1), and diseases which are associated with hemorrhagic tendencies, such as hemophilia, thrombocytopenic purpura, leukemia, and scurvy (10).

On rare occasions, hemopneumothorax occurs suddenly as a dramatic medical emergency with signs of serious internal hemorrhage in the absence of any underlying disease process or chest injury. This is known as spontaneous hemopneumothorax because no obvious pulmonary disease is present prior or subsequent to the hemorrhage into the pleural cavity, if the patient survives. The patient is not incapacitated after recovery if the affected lung has fully expanded, and there is not an excessive amount of residual pleural thickening. Recurrences of pneumothorax or hemopneumothorax may, however, occur (2, 3).

Spontaneous hemopneumothorax is certainly not common; there are about 60 cases in the medical literature (4). It is quite probable, however, that there are a good many cases, both unrecognized and unreported. The mortality rate is relatively high, since in 14 of the reported cases the patient succumbed and was autopsied (5). With the aid of this pathological material, it would seem that the cause of the condition would be easy to establish, but this has not proved to be the case. The etiology remains relatively obscure, in the same way that the cause of "essential hematuria" or certain cases of gastrointestinal bleeding remains obscure, even in the presence of exhaustive pathological studies (6, 7).

Clinically, there is a sudden onset of pain on one side of the chest, generally in an individual under forty, and almost always in a male. The pain may be relieved after a short interval or may persist from several hours to several days, when weakness, dyspnea, and shock may ensue. Abdominal pain and rigidity, rather than chest pain, may be present and confuse the issue. The diagnosis is established by the blood count, which demonstrates anemia; the chest film, which shows a hydropneumothorax; aspiration of blood from the affected side of the thorax.

Except for the gravity of the clinical picture of spontaneous hemopneumothorax and its infrequency, there are many points of similarity between it and spontaneous pneumothorax. Both occur in young adults, are sudden in onset, and are manifested by pain in the chest and dyspnea. Neither is preceded by any notable chest trauma. Minor stresses and strains which produce increased intrathoracic pressure, such as lifting a heavy object, throwing it over the shoulder, coughing, sneezing, and straining are, however, frequently recorded in both conditions.

Spontaneous hemopneumothorax may represent spontaneous pneumothorax with the unusual complication of hemorrhage. The mechanism of this hemorrhage into the pleural cavity has been the subject of considerable controversy. This can be readily understood, since no source of

[1] Published with permission of the Chief Medical Director, Veterans Administration, who assumes no responsibility for the opinions expressed or the conclusions drawn by the author. Accepted for publication in July 1948.

hemorrhage was demonstrated in half of the autopsied cases. Even the origin of the pneumothorax could not be found in most instances, and in three cases no lesions of any sort were discovered in the collapsed lung.

Emphysematous bullae of the collapsed lung were present in 10 cases, but there was rupture of a bulla in only 4 cases. In some of the cases blood was seen oozing from a torn bulla, and it was justifiably assumed by those reporting these cases (2) to be the source of both the air and blood in the pleural cavity. Others have denied that bleeding from a ruptured bulla or bleb could be extensive even though the bulla was highly vascularized, since the pressure in the pulmonary circulation is low (2). It must be recalled, however, that the lungs are also supplied through the bronchial arteries by the systemic circulation, the pressure in which is six times greater than that in the lesser circulation and considerably above that in the pleural cavity.

Pleural adhesions on the affected side of the chest were found in 6 of the 14 cases, and in 5 of these they were torn. They were found to be highly vascular in several of the cases, and probably the source of bleeding. The hemorrhage in such cases is believed to occur from the parietal side of the torn adhesions (2), which is directly supplied by branches of the intercostal vessels. Tearing of adhesions during the induction of artificial pneumothorax in the treatment of tuberculosis is an occasional source of hemorrhage (3), and that these pleural adhesions may be a source of serious pleural bleeding is readily demonstrable during the procedure of pneumonolysis (8). In spontaneous hemopneumothorax, pleural adhesions may be torn when the lung collapses as a result of rupture of a peripherally situated pulmonary bulla or a congenital subpleural bleb (2). On the other hand, muscular exertion or jarring of the thorax is believed by some to cause tearing of pleural adhesions preceding the pneumothorax (5).

The recent work of Macklin and Macklin (9) emphasizes interstitial emphysema of the lung produced by rupture of pulmonary alveoli within the lung as a cause of spontaneous pneumothorax and hemopneumothorax. The rupture of alveoli about vascular sheaths is produced by an increase in the gradient of pressure between the alveoli and the blood vessels within the sheaths. This increased gradient of pressure may be brought about by hyperinflation of certain alveoli and/or a diminished amount of blood within the vascular sheaths of the lung, such as might be due to straining with the glottis closed. After the rupture of the alveoli, air travels along the vascular sheaths to the root of the lung and into the mediastinum. Pneumothorax is produced by rupture of the mediastinal pleura. The air may also travel to the periphery of the lung, producing subpleural blebs which also may rupture to produce pneumothorax. In hemopneumothorax, capillaries at the bases of the ruptured alveoli are also torn, and the blood may escape into the pleural cavity along the pathways followed by the air. This method of producing a hemopneumothorax has been demonstrated by these investigators in animal experiments.

It is apparent that the assumption that spontaneous hemopneumothorax occurs in the complete absence of underlying pulmonary disease is to some extent erroneous. Subpleural pulmonary bullae are the result of obstructive emphysema due either to non-specific inflammatory changes or to the scarring produced by minimal tuberculous lesions. Some of these bullae may be congenital in origin. Pleural adhesions, though very commonly found at autopsy, are, nevertheless, generally the end-result of an inflammatory process of the pleura. Macklin and Macklin postulate an underlying weakness of the alveolar walls which predisposes them to rupture and leads to the development of interstitial emphysema of the lungs and mediastinum. This weakness of the alveolar walls may be produced by the toxins of certain infectious diseases such as influenza, or may be due to inherited constitutional defect.

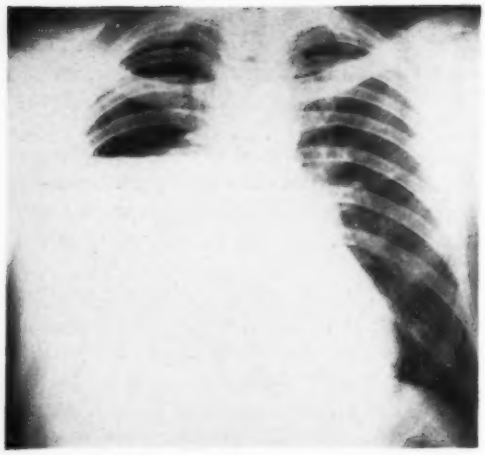


Fig. 1. Initial chest film showing hemopneumothorax.

CASE REPORT

H. M., a 32-year-old white male, an automobile clerk, was admitted to the hospital on Nov. 4, 1947, complaining of pain in the right chest and right shoulder, weakness, pallor, dyspnea, and tachycardia. The pain in the chest and shoulder came on suddenly, six days before admission, while the patient was standing still, and had continued. Dyspnea and tachycardia also occurred at the time of the onset of pain, but weakness and pallor came on later.

There was no history of recent weight loss, cough, or expectoration. There had been no similar episode in the past. The family history was negative except that one aunt had died of tuberculosis when the patient was thirteen years of age.

Physical examination showed the patient to be well nourished, moderately dyspneic, and very pale. Lagging of the right side of the chest on inspiration was noted. There were flatness on percussion at the right base and hyperresonance above this. No breath sounds were audible in the right lung. The heart was deviated to the left, but heart sounds were normal and no murmurs were heard. The blood pressure was 120/60, the pulse 94. The remainder of the physical examination was negative. The admission diagnosis was pleurisy with effusion.

An emergency roentgenogram of the chest showed a right hydropneumothorax (Fig. 1). The fluid in the right pleural cavity extended up to the level of the posterior axillary portion of the right fifth rib. The lower portion of the cardiac silhouette was displaced to the left. The blood count was 1,960,000 red cells, hemoglobin 40 per cent, 11,200 white cells (neutrophils 69, lymphocytes 26, eosinophils 5, basophils 2).

At this point the correct diagnosis of spontaneous hemopneumothorax was suspected, and was confirmed by aspiration of 100 c.c. of dark liquid blood.

A culture of the chest fluid showed no growth, and a smear showed no tubercle bacilli or other organisms. Blood culture was negative. Repeated sputum concentration studies for acid-fast bacilli were negative. Another specimen of the chest fluid was examined for acid-fast bacilli and malignant cells, but none were found.

The patient was treated energetically, and his condition rapidly improved. The right side of his chest was aspirated on each of three successive days, resulting in the total withdrawal of about 2,500 c.c. of fluid. Much smaller amounts were removed on subsequent aspirations. Two transfusions of 500 c.c. of blood were given. A febrile reaction, with temperature up to 102.4°, followed the first transfusion, and the patient had a low-grade fever for the next five days, the highest level being 100° F. He then became afebrile and asymptomatic. On Nov. 10, 1947, the blood count showed 4,170,000 red cells, hemoglobin 78 per cent, 10,400 white cells (neutrophils 82, lymphocytes 16, eosinophils 2).

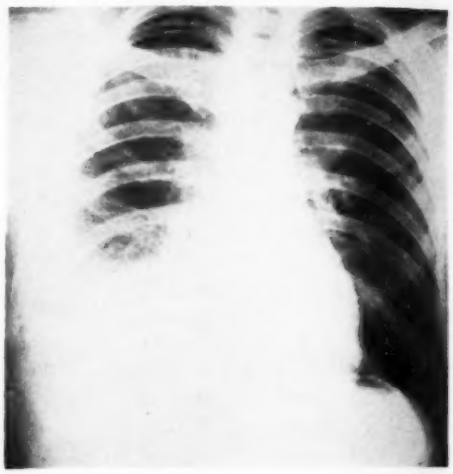


Fig. 2. Film obtained after several chest aspirations. Much of the blood has been removed, and the right lung has almost completely re-expanded. A small amount of air is still present in the apical portion of the right pleural cavity.

Serial films of the chest (Figs. 2 and 3) showed progressive re-expansion of the right lung and rapid decrease in the amount of fluid in the right pleural cavity. On Dec. 30, 1947, there was noted residual pleural thickening at the periphery of the right lower pleural cavity and in the right costophrenic sinus. Both lungs were completely re-expanded and there was no roentgen evidence of pulmonary bullae.

In conclusion, it is evident that the symptom-complex of spontaneous hemopneumothorax, though clear-cut in its manifestations, presenting anemia due to hemorrhage, fluid and air in the pleural

cavity demonstrable on x-ray examination, and bloody fluid on aspiration, has no uniform mode of production.

Pneumothorax may occur due to rupture of a subpleural emphysematous bulla or a congenital subpleural bleb. It may also be a complication of interstitial emphysema of the lung and mediastinum following rupture of pulmonary alveoli, with leakage of air through the visceral or mediastinal pleura into the pleural cavity.

Hemorrhage may occur from the rupture of highly vascular pulmonary bullae, from pleural adhesions torn by muscular exertion, jarring of the chest, or spontaneous pneumothorax. It may also be due to rupture of pulmonary capillaries at the bases of torn alveoli during the production of interstitial emphysema of the lung, with blood following the same pathway as the air into the pleural cavity.

Since the mechanism of hemopneumothorax is often not ascertained at autopsy, it is obvious that its determination during life would be even more difficult, if not impossible. The various possible causes of spontaneous hemopneumothorax illustrate the complexity of the problem.

Veterans Administration Hospital
Montgomery 10, Ala.

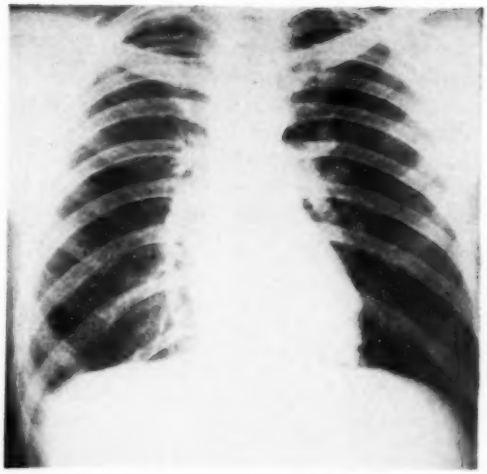


Fig. 3. Complete re-expansion of right lung. Slight pleural thickening remains at the periphery of the right pleural cavity.

REFERENCES

1. RAWSON, A. J., AND COCKE, J. A.: Infarction of an Entire Pulmonary Lobe with Subsequent Aseptic Softening Causing Sterile Hemopneumothorax. Am. J. M. Sc. **214:** 520–524, November 1947.

2. HARTZELL, H. C.: Spontaneous Hemopneumothorax. Report of Three Cases and a Review of Literature. Ann. Int. Med. **17:** 496–510, September 1942.

3. WARING, J. J.: Spontaneous Hemopneumothorax. Clinics **4:** 940–958, December 1945.

4. VAN DER MEER, R.: Spontaneous Hemopneumothorax. Case Report. Am. Rev. Tuberc. **54:** 283–286, September 1946.

5. HELWIG, F. C., AND SCHMIDT, E. C. H.: Fatal Spontaneous Hemopneumothorax: Review of Literature and Report of Case. Ann. Int. Med. **26:** 608–617, April 1947.

6. CROHN, B. B., MARSHAK, R. H., AND GALINSKY, D.: Repeated Gastroduodenal Hemorrhages Without Discoverable Explanation. Gastroenterology **10:** 120–128, January 1948.

7. SPORER, A., AND POLLOCK, R.: Renal Varix. J. Urol. **58:** 424–427, December, 1947.

8. TUCKER, W. B.: Artificial Pneumothorax and Other Collapse Therapy in Pulmonary Tuberculosis. Clinics **4:** 906–928, December 1945.

9. MACKLIN, M. T., AND MACKLIN, C. C.: Malignant Interstitial Emphysema of the Lungs and Mediastinum as an Important Occult Complication in Many Respiratory Diseases and Other Conditions: An Interpretation of the Clinical Literature in the Light of Laboratory Experiment. Medicine **23:** 281–358, December 1944.

10. CAFFEY, J.: Pediatric X-Ray Diagnosis. Chicago, Year Book Publishers, Inc., 1945, p. 296.

SUMARIO

Hemoneumotórax Espontáneo: Consideraciones Etiológicas e Historia Clínica

El síndrome de neumotórax espontáneo, aunque bien definido en sus manifestaciones comprendiendo anemia debida a hemorragia, líquido y gas en la cavidad pleural, distinguibles al examen roentgenológico, y líquido sanguinolento al aspirar, no reconoce una patogenia uniforme.

El neumotórax puede sobrevenir a consecuencia de la rotura de una flictena enfisematosa subpleural o subpleural congénita. Puede representar una complicación del enfisema intersticial del pulmón y el mediastino, consecutiva a rotura de alvéolos pulmonares, con escape de aire a través de la pleura visceral o mediastínica a la cavidad pleural.

La hemorragia puede proceder de la rotura de flictenas pulmonares muy vascu-

larizadas, del desgarre de adherencias pleurales por el ejercicio muscular, de la conmoción del tórax o del neumotórax espontáneo. Puede también deberse a rotura de los capilares pulmonares en las bases de los alvéolos desgarrados durante la producción del enfisema intersticial del pulmón, siguiendo la sangre la misma vía que el aire a la cavidad pleural. Ni aun en la autopsia se descubre a veces el mecanismo exacto.

En el caso comunicado, indicaron el diagnóstico los hallazgos radiológicos y la hematometría, confirmándolo la aspiración de sangre, del tórax. Tras aspiraciones repetidas y la transfusión, el enfermo mejoró, obteniéndose con el tiempo la reexpansión total del pulmón.

Arteriovenous Aneurysm of the Lung

A Case Report[1]

ALICE ETTINGER, M.D.,[2] HEINZ MAGENDANTZ, M.D.,[3] and EDWARD A. RUSSO, M.D.[4]

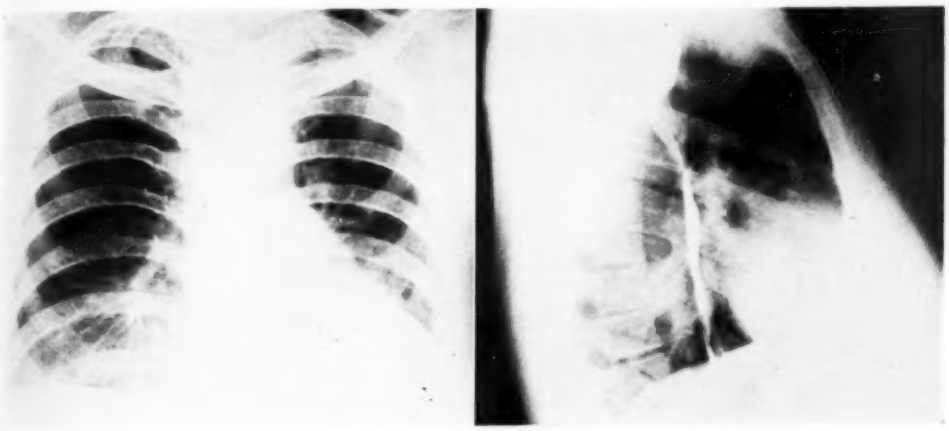
Boston, Mass.

IN THE PAST FEW years there have appeared in the literature an increasing number of reports of arteriovenous aneurysm of the lung (1–20). Most of the cases were diagnosed at operation, but in a small number the diagnosis was established preoperatively on the basis of the clinical or roentgen findings. Untreated, the lesion as a rule leads to death by massive hemoptysis. It is amenable, however, to surgical treatment, and its prompt recognition is therefore of the utmost importance.

The case to be reported here is of particular interest because of repeated failure to recognize the lesion in a considerable number of hospital admissions elsewhere.

Mrs. D. T., a 34-year-old housewife, was admitted to the Joseph H. Pratt Diagnostic Hospital on Feb. 4, 1947, complaining of intermittent hemoptysis over a period of ten years, during which she had been repeatedly hospitalized.

Her first hospital admission, elsewhere, had been on Aug. 26, 1937, following a sudden massive hemoptysis, preceded for several days by a mild cough productive of slightly blood-streaked sputum. On this occasion the patient was acutely ill and cyanotic, and numerous râles were heard throughout the chest. She was placed in an oxygen tent, where two days later she was delivered of a full-term stillborn child. Laboratory studies yielded no significant findings. X-ray examination of the chest was suggestive of a tuberculous infection, and subsequent films showed a shadow at the base of the left lung consistent with consolidation.



Figs. 1 and 2. Postero-anterior and lateral chest films. In the former, the characteristic lobulated shadow is obscured by the heart shadow. Note displacement of heart to the left as the result of pleural pull secondary to previous pneumothorax.

In the lateral view broad bands are seen connecting a lobulated shadow in the left lower lobe with the hilar region.

Following her discharge from the hospital, Sept. 15, 1937, the patient spent nine months in a sanatorium, where an attempt was made to determine the significance of the pulmonary shadow. During this period all laboratory studies were negative. The shadow supposedly resolved to a considerable extent.

Again pregnant, the patient was rehospitalized in February 1939, because of recurrent hemoptysis

[1] Accepted for publication in July 1948.

[2] Roentgenologist-in-Chief, Joseph H. Pratt Diagnostic Hospital, Boston, Mass.; Assistant Professor of Radiology, Tufts College Medical School.

[3] Assistant Professor of Medicine, Tufts College Medical School; Physician, Joseph H. Pratt Diagnostic Hospital.

[4] Resident in Medicine, Joseph H. Pratt Diagnostic Hospital.

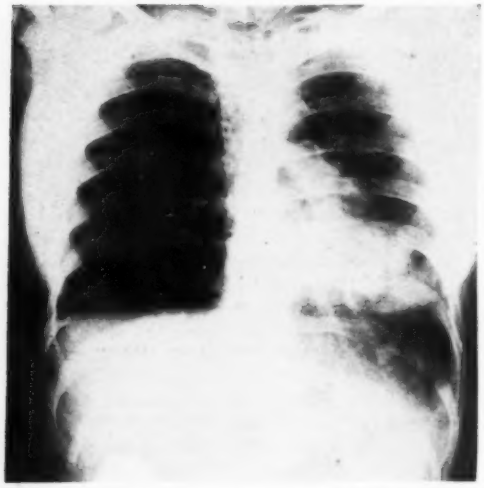


Fig. 3. Bucky film, showing lobulated shadow to best advantage behind the heart shadow. In addition, two small round shadows are seen on the same side in a more basal location.

coming on after the development of a transient right hemiplegia. Again all laboratory findings were normal, and the cause of the bleeding remained obscure.

A third hemoptysis, a month later, necessitated readmission to the hospital. This time tubercle bacilli were found in a single specimen of sputum and the patient was transferred to a sanatorium. The lung shadow previously observed had increased in size, and artificial pneumothorax was instituted, being maintained for the next two years. The second pregnancy terminated uneventfully.

A subsequent hospital admission was necessary because of pelvic inflammatory disease, in the course of which a fourth hemoptysis occurred. During the ensuing four years, the patient received repeated fluoroscopic check-ups. Throughout this period she remained essentially asymptomatic and was able to do her housework.

On Oct. 4, 1946, a fifth severe hemoptysis led to rehospitalization. X-ray examination of the chest now showed a structureless shadow nearly obliterating the left lung markings, which was thought for the most part to represent fluid. Bronchographic studies at a subsequent date led to a diagnosis of probable atelectasis of the left lower lobe. It was at this time that the case was referred to the Pratt Diagnostic Hospital for further investigation.

On admission, the patient was in obvious distress. The outstanding findings on physical examination were dyspnea, marked cyanosis of the skin, mucous membranes, and nail beds, pronounced clubbing of the fingers and toes, fine moist râles at both lung bases, a grade I apical systolic murmur, and telangiectasia of the nasal mucosa. Later, when the diagnosis was suspected, it was possible to distinguish a bruit in the left posterior axillary line, independent of the apical murmur.

X-ray examination of the chest on Feb. 5, 1947 (Fig. 1) was reported as follows: "The right diaphragm is elevated. The right costophrenic angle is obliterated. The left diaphragm is poorly delineated. There are a few strands of increased density in the periphery of the left lower lung field. The left costophrenic angle is not well seen. The heart and mediastinum are displaced into the left chest cavity. The lateral view (Fig. 2) shows a well defined shadow close to the left hilus but no pressure upon the esophagus. There seems to be a band-like connection between this shadow and the hilar region. A Bucky film (Fig. 3) reveals a lobulated mass behind the heart shadow, measuring 4 × 5 cm. In addition, there are two small, poorly defined round masses overlying the tenth rib posteriorly on the left side. The heart is normal in size and shape.

"*Diagnosis:* The findings are rather characteristic of arteriovenous aneurysm of the lung. The mediastinal displacement to the left is the result of pleural adhesions secondary to the previous pneumothorax.

LABORATORY STUDIES: PREOPERATIVE AND POSTOPERATIVE*

Laboratory Studies	Preoperative		Postoperative		
	Feb. 5, 1947	Feb. 11, 1947	March 3, 1947	June 9, 1947	June 10, 1947
Hgb	115% (17.9 gm.)	...	96% (13.9 gm.)	80% (12.5 gm.)	...
Red cells	6,320,000	...	4,110,000	4,490,000	...
Hematocrit	61%	...	...	44%	...
White cells	8,500	...	10,500	6,500	...
Total circulating blood volume	...	8,766 c.c.	...	...	5,375 c.c.
Plasma volume	...	3,429 c.c.	...	...	3,010 c.c.
Red cell volume	...	5,337 c.c.	...	...	2,365 c.c.
Circulation time	15 and 16 sec.	...	...	...	...
Venous pressure	56 mm.	...	...	...	...
Arterial O_2 content at rest	...	19.9 vol. %	...	...	...
Arterial O_2 capacity at rest	...	24.9 vol. %	...	...	...
O_2 saturation at rest	...	79.9%	...	...	...

* Operation performed Feb. 26, 1947.

Fig. 4. Laminagram bringing out basal opacities to better advantage. (The area of marked contrast represents a residue of a previous lipiodol injection.)

This displacement causes the mass shadow of the aneurysm in the ordinary postero-anterior chest film to be hidden behind the cardiac shadow. The heart size is normal, as is usual in arteriovenous aneurysm of the pulmonary circulation."

The diagnosis having been suggested by the roentgen findings, additional laboratory and x-ray studies were carried out. Laminagraphy (Fig. 4) showed the two small round shadows in the lower left lung field to better advantage than the Bucky film. Spot films taken with the patient performing the Valsalva test (Fig. 5)[5] showed a reduction in the shadow to about half its original diameter. The laboratory findings are given in the accompanying table. Hemoglobin and red blood cells were greatly increased. There was also an increase in the total circulating blood volume, attributable to an increased red cell volume rather than to the plasma. Circulation time and venous pressure were normal. The arterial hemoglobin oxygen saturation was markedly diminished at rest.

The bruit heard in the left posterior axillary line after the diagnosis of arteriovenous aneurysm was suspected was shown (Fig. 7) to occur between the first and second heart sounds.

A left lower lobectomy was performed on Feb. 26, 1947, by Dr. R. H. Betts.

[5] Lindgren, in 1946, suggested that the increase in intrapulmonony pressure on performance of the Valsalva maneuver may result in a marked decrease of an aneurysmal shadow, thus proving the vascular nature of the lesion (12).

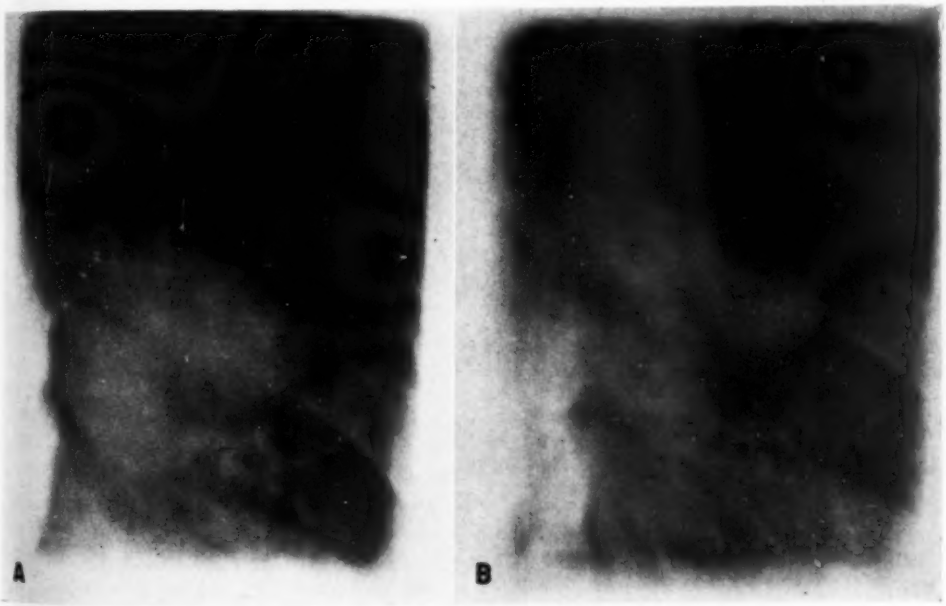


Fig. 5. A. Close-up of shadow in quiet respiration. B. Same during Valsalva test. Note striking decrease of size of mass.

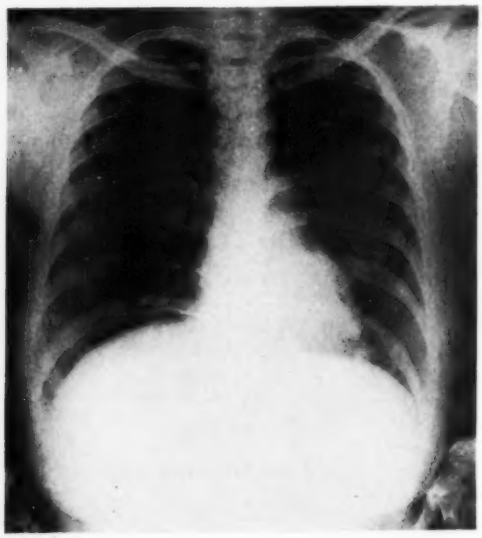


Fig. 6. Film made on July 1, 1938 (procured from another hospital) before induction of pneumothorax. A poorly defined mass is present in the left lower lobe.

Operative Findings (Fig. 8): The lung was adherent throughout to the chest wall and diaphragm. A persistent bleeder was encountered in the cupola, and the bleeding was controlled with difficulty. The interlobar fissure line was fused, but the vessels were freely isolated. The pulmonary artery was twice its normal size and the inferior pulmonary vein about two and a half times its normal size. The lingular branches of the artery were small and of normal caliber. The angioma itself was located in the posterior basilar segment, and had a lobulated appearance. At various times, with mild manual pressure, a thrill could be felt.

Pathological Findings (Fig. 9): The surface of the excised lobe showed shaggy fibrous adhesions. The parenchyma appeared collapsed. On section, the tissue was pale gray and of spongy consistency. The main artery was found to lead into a cavernous and multilocular area, measuring 3.5 cm. in greatest diameter. Dilatation began 3.3 cm. from the cut end of the artery and the lobules showed numerous anastomosing communications. The entire aneurysm lay just beneath the anteromedial pleural surface, on a level with the inferior main segmental bronchi. The vein accompanying the main artery showed dilated anastomosing channels, less marked than in the case of the artery, but definitely communicating with the arterial channels. Finer vessels appeared at the periphery of the aneurysm, apparently plugged with clot. A slender, uninvolved tributary of the vein passed immediately beneath the aneurysm, but did not communicate with it. This tributary followed along the path of an uninvolved arterial branch leading off from the dilated artery

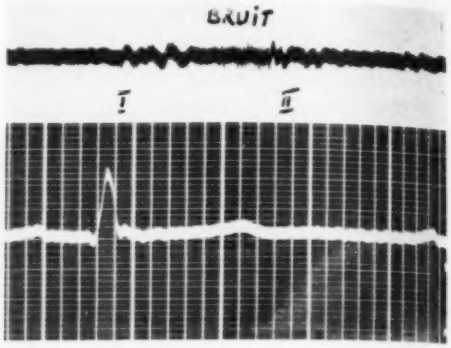


Fig. 7. Stethogram taken with medium bell over posterior axillary line, at level of 6th rib, in right recumbent position.

and supplying the anterior basal segment. The walls of the segmental bronchi showed minimal thickening.

Microscopic Diagnosis: Arteriovenous aneurysm.

Postoperative Course: There was immediate improvement in the oxygenation of the peripheral circulation. The red blood count and hemoglobin dropped to 4,110,000 and 13.9 gm., respectively, from the original 6,320,000 and 17.9 gm. The mucous membranes and nail beds became pink. The total blood volume fell from 8,766 c.c. to 5,375 c.c. due chiefly to diminution of the red blood cells. The hematocrit reading fell from 61 to 44 per cent. After an uneventful convalescence, the patient was discharged on the fifteenth postoperative day. She was seen again on Oct. 23, 1947, and stated that she felt fine and had gained twenty pounds. Her cyanosis had completely disappeared, and though there was still some clubbing, it was less than preoperatively. In February 1948, the patient gave birth to a healthy child.

DISCUSSION OF ROENTGEN DIAGNOSIS

The question arises whether the roentgen findings in arteriovenous aneurysm of the lung are of such nature as to suggest the diagnosis. It is believed that in the majority of cases they are. A lobulated mass connected with the hilus by band-like vascular shadows is highly suggestive of arteriovenous aneurysm. In order to prove the vascular nature of the shadow, several approaches are possible. One would expect pulsations to be demonstrable fluoroscopically, but this observation has been reported in only 5 cases. Apparently the size of the feeder vessel is a determining factor, these vessels being very large in the cases (2, 4, 7, 11) in which pulsations were

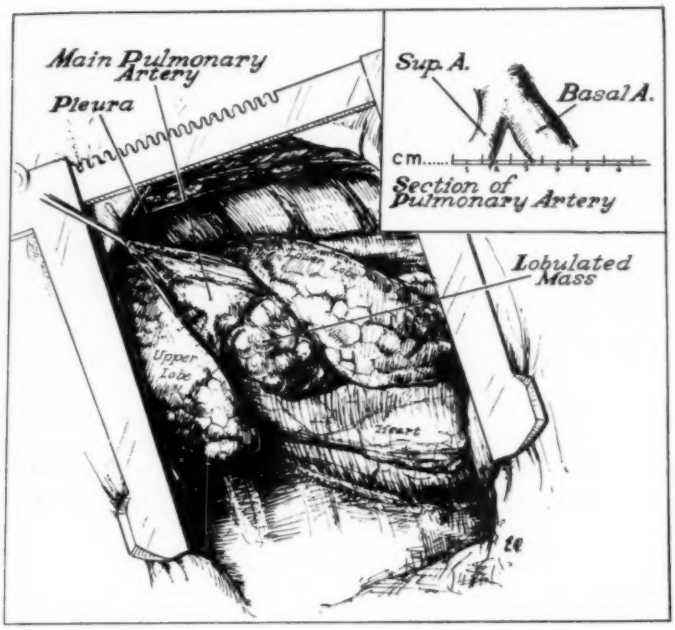


Fig. 8. Operative findings.

observed. In one of Goldman's cases (6), pulsations were seen also on kymographic study. In most reports no accurate statement has been made as to the presence or absence of pulsations, probably because the diagnosis was not established roentgenologically. Laminagraphy is frequently helpful in demonstrating the connection of the tumor-like shadow with the hilar region. Angiography would seem, of course, to be the most conclusive means of establishing the vascular nature of the lesion, but it may be dangerous, and at least one case has been recorded (18) in which death followed its use.

Lindgren (12) was the first to point out the feasibility of using the change of intrathoracic pressure by the Valsalva test as a means of determining the vascular nature of the lesion. Since his report, this test has been used by Makler and Zion (14) and in our case. It is an exceedingly valuable diagnostic measure and obviates the need for angiography in those cases in which a typical lobulated shadow gives a characteristic response.

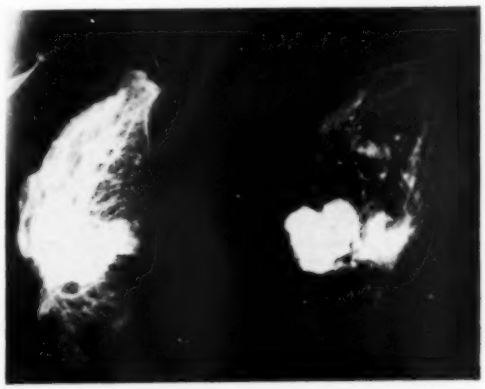


Fig. 9. Operative specimen injected with diodrast. (Courtesy of Dr. R. H. Betts, New England Deaconess Hospital, Boston, Mass.)

In a review of the published cases, plain films were found to be characteristic in all but two (12,[6] 17). In these latter cases, accompanying hemorrhage obscured the picture. The diagnosis is further supported by the demonstration of small round shadows in other parts of the lung. These were present in more than half the published

[6] Third case.

cases. They are due to small additional hemangiomatous malformations.

The chief condition to be considered in the differential diagnosis, so far as the roentgen picture is concerned, is tumor of the lung, but the clinical syndrome which is associated with arteriovenous aneurysm is not seen with tumors. Multiple round shadows in the lung, such as have been described in polycythemia vera (21) should offer no diagnostic problem, since they are not vascular in character and should, therefore, show no change in size with the Valsalva test.

CLINICAL DIAGNOSIS

The syndrome of polycythemia, cyanosis, clubbing of the fingers and toes, repeated hemoptyses, breathlessness, faintness, dizziness and weakness, and a distant bruit or hum in the chest, associated with a pulmonary shadow and roentgen evidence of small hemangiomata in the upper respiratory tract should arouse suspicion of arteriovenous aneurysm and lead to special x-ray studies and blood volume determinations.

An arteriovenous aneurysm in the peripheral circulation causes cardiac dilatation and signs of congestive heart failure. The larger the shunt and the nearer to the heart, the more serious the consequence for the circulation. An arteriovenous aneurysm in the lung, however, does not lead to cardiac enlargement, as seen in most of the reported cases and in our own. The strikingly increased circulating blood volume is presumably related to the cyanosis, which, in turn, is explained by the shunt of venous blood from the pulmonary artery directly into the pulmonary vein and, consequently, into the peripheral arterial system, without benefit of oxygenization. The increased total volume of the circulating blood is due to an increase of the red blood cells, but not of the plasma, as recently pointed out by Maier and collaborators (13). In our patient the red blood cell volume fell markedly after operation, accounting thus for most of the diminution in total circulatory blood volume.

Clubbing is a common finding in congenital heart disease with impaired oxygen content of the peripheral arterial blood. The markedly reduced oxygen saturation of the arterial blood in our case—only 79.9 per cent—corresponds to similar findings in other cases, and can easily be explained by the veno-arterial shunt in the pulmonary aneurysm. A shift of the electrical axis of the electrocardiogram to the left, postoperatively, was noted, which may be related to the disappearance of the erythrocythemia or the lessened burden on the right ventricle.

In analyzing the salient features of this rare congenital vascular disease, one must conclude that the cardinal symptoms and signs are distinct from any other clinical picture and that, therefore, the diagnosis can readily be made if one is familiar with the syndrome. On the other hand, superficial examination may lead to confusion with other conditions that simulate parts of the syndrome.

The repeated hemoptyses may lead to a suspicion of bronchiectasis, which would be further strengthened by the clubbing of the fingers, but the cyanosis and the increased red cell count and volume should exclude this diagnosis, as should, also the x-ray findings.

Mitral stenosis may need to be briefly considered, since it may lead to repeated hemoptyses and cyanosis, and perhaps occasional clubbing. In that condition, however, the characteristic apical rumbling murmur is rarely absent, and in cases with marked pulmonary congestion and hemoptysis the x-ray silhouette of the heart is almost always typical.

The cyanotic group of congenital heart diseases, as pulmonary stenosis, tetralogy of Fallot, and the pulmonary dilatation of Eisenmenger's complex with an overriding aorta, may have to be ruled out, since they are associated with cyanosis, dyspnea, clubbing, and dizziness, but they do not give rise to hemoptysis and often have distinct x-ray and electrocardiographic signs and physical findings.

A further finding suggestive of arterio-

venous aneurysm is the presence of multiple small hemangiomata in the nose, mouth, and upper respiratory tract, as reported by Rodes (22). Injection of small amounts of ether into the antecubital vein, as in circulation time studies, may produce a peculiar crawling sensation in the skin, as noticed in congenital septal defects or an overriding aorta. One may also encounter severe headaches and momentary narcosis during this test. The procedure was not attempted in the case reported here, since its value in congenital heart disease was not known to us at the time. Moreover, it is dangerous in such cases.

SUMMARY

A case of pulmonary arteriovenous aneurysm is reported, in which the diagnosis was made roentgenologically, and surgical cure was achieved by lobectomy. The roentgenologic means of establishing the diagnosis are discussed, and the value of the Valsalva test is emphasized. The clinical and laboratory data are presented, and the conclusion reached that knowledge of the clinical syndrome, together with the laboratory findings and x-ray studies, permits one to make this diagnosis with a high degree of accuracy, as a rule without the aid of more complicated procedures, such as angiography.

REFERENCES

1. ADAMS, W. E., THORNTON, T. F., JR., AND EICHELBERGER, L.: Cavernous Hemangioma of the Lung. Report of a Case with Successful Treatment by Pneumonectomy. Arch. Surg. **49:** 51–58, July 1944.

2. BEIERWALTES, W. H., AND BYRON, F. X.: Pulmonary Arteriovenous Aneurysm with Secondary Polycythemia. Report of First Case Treated by Lobectomy. J. A. M. A. **134:** 1069–1072, July 26, 1947.

3. BISGARD, J. D.: Discussion of Maier, *et al.* (13).

4. BURCHELL, H. B., AND CLAGETT, O. T.: Clinical Syndrome Associated with Pulmonary Arteriovenous Fistulas. Am. Heart J. **34:** 151–162, August 1947.

5. BYRON, F. X.: Discussion of Maier, *et al.* (13).

6. GOLDMAN, A.: Cavernous Hemangioma of the Lung; Secondary Polycythemia. Dis. of Chest **9:** 479–486, November-December 1943.

7. GOLDMAN, A.: Pulmonary Arteriovenous Fistula with Secondary Polycythemia Occurring in Two Brothers; Cure by Pneumonectomy. J. Lab. & Clin. Med. **32:** 330–331, March 1947.

8. HEPBURN, J., AND DAUPHINEE, J. A.: Successful Removal of Hemangioma of Lung Followed by Disappearance of Polycythemia. Am. J. M. Sc. **204:** 681–685, November 1942.

9. JANES, R. M.: Discussion of Jones and Thompson (11).

10. JANES, R. M.: Multiple Cavernous Hemangioma of the Lungs Successfully Treated by Local Resection of the Tumours. Brit. J. Surg. **31:** 270–272, January 1944.

11. JONES, J. C., AND THOMPSON, W. P.: Arteriovenous Fistula of the Lung. J. Thoracic Surg. **13:** 357–373, October 1944.

12. LINDGREN, E.: Roentgen Diagnosis of Arteriovenous Aneurysm of the Lung. Acta radiol. **27:** 585–600, 1946.

13. MAIER, H. C., HIMMELSTEIN, A., RILEY, R. L., AND BUNIN, J. J.: Arteriovenous Fistula of the Lung. J. Thoracic Surg. **17:** 13–22, February 1948.

14. MAKLER, P. T., AND ZION, D.: Multiple Pulmonary Hemangiomata. Am. J. M. Sc. **211:** 261–266, March 1946.

15. SAMSON, P. C.: Discussion of Maier, *et al.* (13).

16. SHEFTS, L. M.: Discussion of Maier, *et al.* (13).

17. SHENSTONE, N. S.: Experiences with Total Pneumonectomy. J. Thoracic Surg. **11:** 405–413 April 1942.

18. SISSON, J. H., MURPHY, G. E., AND NEWMAN, E. V.: Multiple Congenital Arteriovenous Aneurysms in the Pulmonary Circulation. Bull Johns Hopkins Hosp. **76:** 93–111, March 1945.

19. SMITH, H. L., AND HORTON, B. T.: Arteriovenous Fistula of Lung Associated with Polycythemia Vera. Am. Heart J. **18:** 589–592, November 1939.

20. SWEET, R. H.: Discussion of Maier, *et al.* (13).

21. HIRSCH, I. S.: Pulmonary Changes in Polycythemia Vera. Radiology **26:** 469–473, April 1936.

22. RODES, C. B.: Cavernous Hemangioma of Lung with Secondary Polycythemia. J. A. M. A. **110:** 1914–1915, June 4, 1938.

72 Addington Road
Brookline, Mass.

SUMARIO

Aneurisma Arteriovenoso del Pulmón. Observación

En el caso de aneurisma arteriovenoso del pulmón, comunicado, el diagnóstico se hizo con los rayos X y se obtuvo la curación quirúrgica con la lobectomía. Clínicamente, debe sospecharse tal estado en presencia de hemorragias repetidas, policitemia, cianosis, dedos hipocráticos, disnea, desmayos, vértigo, debilidad y ruido remoto en el tórax. Radiológicamente, la presencia de una tumefacción lobulada unida al hilio por sombras vasculares parecidas a franjas es muy indicativa de aneurisma arteriovenoso. La disminución del tamaño de la sombra al ejecutar la prueba de Valsalva confirmará la naturaleza vascular de la lesión.

Volvulus of the Sigmoid: A New Radiologic Sign[1]

DR. M. ARIAS BELLINI

Montevideo, Uruguay

According to statistics, volvulus of the sigmoid is responsible for 45 per cent of obstructions in the large intestine; it is therefore one of the most frequent causes of intestinal obstruction in general. Volvulus of the sigmoid is the result of torsion of the sigmoid colon around its mesenteric axis. The pivot of this torsion is the rectosigmoid, the anatomic characteristics of which permit the twisting of the mobile pelvic colon.

With the growth and development of the individual, certain changes take place in the pelvic colon. The wide mesocolon of the fetus and newborn infant becomes the narrow mesocolon of the adult, while the coil elongates. Frequently, however, the evolution of these two structures is not parallel, and the length of the coil may become excessive in relation to the mesentery. Under these conditions torsion is very probable.

To the anatomic conditions predisposing to volvulus are to be added certain pathological processes which may affect either the mesocolon or the sigmoid coil. The base of the mesocolon, normally small, may be still further reduced by inflammatory processes (a retractile mesosigmoiditis), being transformed into a mere pedicle. A megacolon may also involve the coil and facilitate torsion.

Having outlined thus briefly the anatomical and pathological causes that contribute to the frequency of torsion of the sigmoid, we may proceed to a discussion of the radiological signs that aid in its diagnosis. The study of a case, from this point of view, begins with two roentgenograms, one with the patient standing and the other in the supine position. A detailed study of these two films sometimes permits a diagnosis without resort to the barium enema.

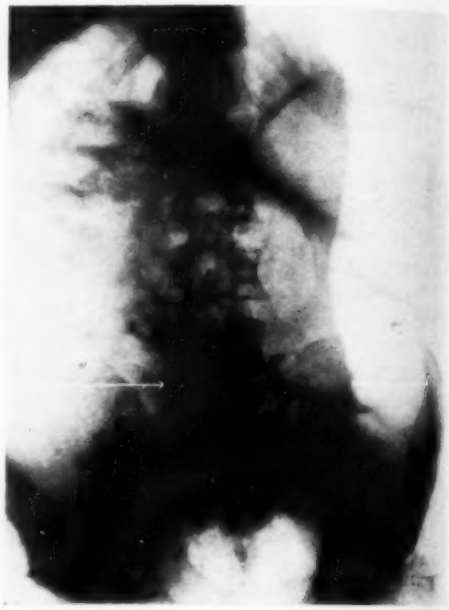


Fig. 1. Occlusion due to a sigmoid neoplasm, with a competent ileocecal valve. The cecum and ascending colon are greatly expanded, while the distention in the descending colon (left) is much less.

RADIOLOGIC SIGNS OF SIGMOID VOLVULUS

In any case of volvulus we have (*a*) a strangulation of an intestinal coil and above this (*b*) an obstruction. In a particular case, the picture will depend upon the competence of the ileocecal valve. With an incompetent valve, we have what is regarded as the typical image of sigmoid volvulus, a huge arched coil with a uniform and ample connection between the two sides of the arch. At first, it may seem as if this coil represents a distention of the whole colon, such as can be seen roentgenographically in cases of sigmoid cancer. When we study the character of the coil, however, we find that it is a segment of the colon only, distended as a result of mechanical obstruction.

[1] Presented before the Radiological Society, Montevideo, May 1948. Accepted for publication in June 1948.

There is one sign which we have found useful in arriving at a correct diagnosis in these cases. In the "hermetically shut coil" in the presence of a neoplasm, the diameter of the right-hand portion is larger than that of the left-hand portion (Fig. 1). The explanation of this is clear, if we consider that the former represents the cecum and ascending colon, classically known to be very distensible, while the left-hand

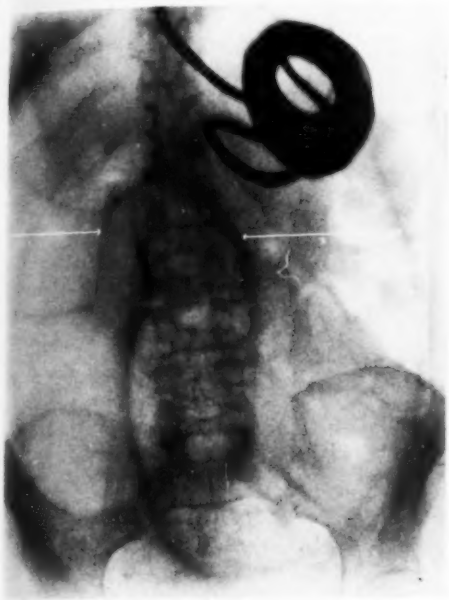


Fig. 2. Typical picture of sigmoid volvulus. The two sides of the arch formed by the distended coil are of uniform diameter with ample communication between them. This characteristic finding excludes a diagnosis of sigmoid neoplasm.

segment is formed by the more rigid descending colon. In cases of sigmoid volvulus (Fig. 2), on the other hand, the two parts of the distended coil show a uniform expansion and diameter, which is logical in segments of a single coil. This sign is of great differential value, though we have seen no mention of it in any of the various texts on this subject.

A competent sphincter complicates the radiologic picture (Fig. 3) in sigmoid volvulus, because in that event we observe a double obstruction: (*a*) in the sigmoid coil, with the characters already described,

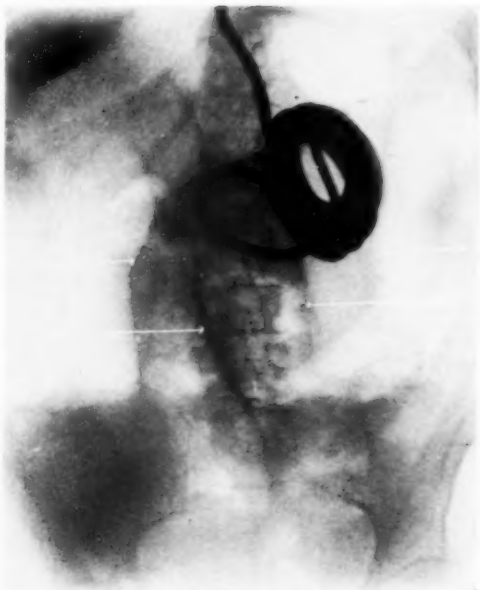


Fig. 3. Sigmoid volvulus with competent ileocecal valve. A double occlusion is seen: of the twisted coil and of the segment of colon between the sigmoid and the ileocecal valve. The image of the twisted sigmoid coil is partially superimposed upon the rest of the distended colon. The cecum and ascending colon (on the right) are also seen, more dilated than the descending colon.

above the square formed by the colon and; (*b*) in the expanded colon, with right and left segments of different diameter. Mondor points out that the radiologic picture is quite confusing in such a case, but the sign that we have described has always suggested the diagnosis and aided in distinguishing radiologically the type of colic occlusion.

THE BARIUM ENEMA

We may now mention briefly the ways in which the barium enema may assist in the diagnosis of volvulus. The barium may either be detained in the rectosigmoid region and form what is known as "snake's head" (Fig. 4) or it may pass the point of obstruction and fill the distended coil. In this latter case, the barium cannot be expelled (Fig. 5). There is no relation whatever between the degree of torsion and the penetration of the barium. We have seen filling of the coil in volvulus of 360 degrees.

Fig. 4. The barium may be retained in the recto-sigmoid region and form what is known as a "snake's head."

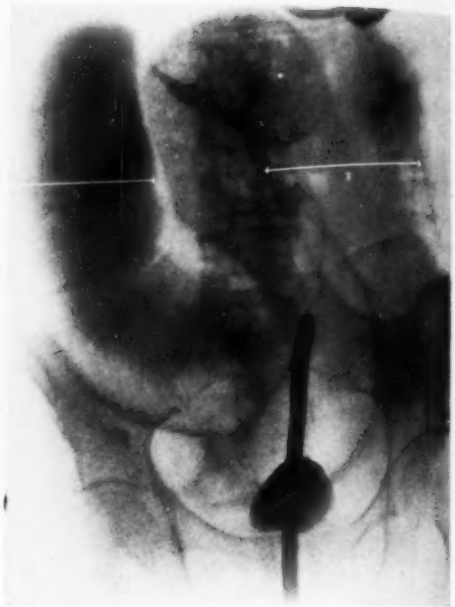


Fig. 5. Barium enema study. The barium has passed the point of obstruction and cannot be expelled. The two segments have a uniform diameter.

SUMMARY

The radiologic picture characteristic of volvulus of the sigmoid is described. A new sign is pointed out which is considered of great value, serving to distinguish this type of colic obstruction from that due to any other cause. The distinctive feature consists in the uniform diameter of both sides of the arch produced by the distended sigmoid coil.

Luis B. Cavia 2622
Montevideo, Uruguay

REFERENCES

DEL CAMPO, J. A.: Abdomen agudo. El Ateneo; Montevideo: Ed. Científico de Sindicato Médico del Uruguay, 1940.

MONDOR, H., AND PORCHER, R.: Radio-diag. urgents. Abdomen. Paris, Masson & Cie, 1943.

PIQUINELA, J. A.: El esfinter ileocecal. Arch. urug. de med., cir. y especialid. **29:** 541–551, 1946.

PRAT, D.: Ileo. Oclusión y obstrucción intestinal. An. Fac. de med., Montevideo **16:** 49; 321; 471, 1931.

ROGLIA, J. L., LORENZO y LOZADA, H., and ZANZI, L.: An. d. ateneo de le clín. quir. **12:** 7, February 1946.

SANTE, L. R.: Intestinal Obstruction. Am. J. Roentgenol. **34:** 744–753, December 1935.

SUMARIO

Ileo de la S Ilíaca: Nuevo Signo Radiológico

El estudio radiológico del vólvulo del sigmoide ofrece un conjunto de signos que permite efectuar un diagnóstico positivo.

El autor señala un nuevo signo que le otorga a la imágen del vólvulo del sigmoide características que la individualizan contribuyendo así a facilitar el diagnóstico diferencial con las otras oclusiones del colon. La característica distintíva del signo consiste en el diámetro uniforme de ambos lados del arco formado por el asa sigmoidea distendida.

Cauda Equina Syndrome Due to Silent Rectal Carcinoma[1]

ROBERT J. GROSS, M.D.

Lyons, N. J.

THE EARLY MANIFESTATIONS of rectal carcinoma are frequently insidious in onset, with symptoms not directly referable to the rectum. Digital, proctoscopic and roentgen studies may fail to disclose the presence of a rectal tumor, unless these procedures are repeated (1). Anemia, weakness, weight loss, and ill-defined, non-localized bowel distress are the most common symptoms of these silent lesions. Even these complaints are not invariably present, as in the case to be described.

Rectal carcinoma does not often metastasize to bone. Mayo and Schlicke found skeletal metastasis in only 1.2 per cent of a group of cases of carcinoma of the colon (2). There are no precise figures on the incidence of sacral involvement, but patients with advanced rectal carcinoma occasionally show evidence of direct extension to the sacrum and sacral nerve roots (3). The case to be presented is interesting in that the initial presenting problem was a cauda equina syndrome. The primary rectal carcinoma remained silent and unsuspected for a considerable period of time. In retrospect it seems that digital or proctoscopic examination should have led to a correct diagnosis.

CASE REPORT

M. R., a 43-year-old white male, was admitted to the hospital in March 1947, on account of pain in the right gluteal region. His father had died of gastric carcinoma. The patient had been in excellent health until four months before admission, at which time he experienced a sudden onset of right lumbar pain. A month later he discovered a lump in the right gluteal region, which rapidly increased in size. Gradually motor power in the right leg was lost and difficulty in urination and defecation developed. The lump was exceedingly tender, and there was a burning sensation in the right perineal region. No history of blood in the stools was elicited. No significant weight loss occurred.

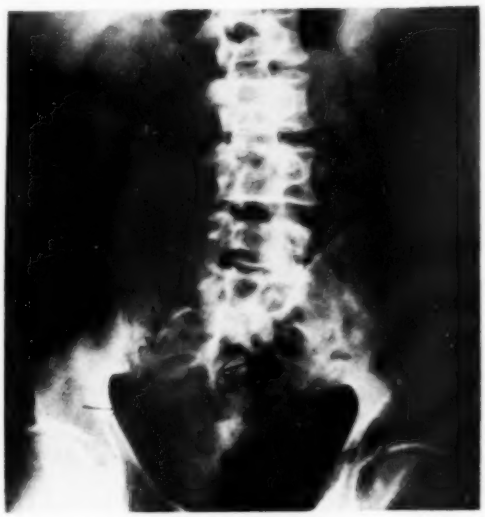


Fig. 1. Roentgenogram of the pelvis showing large area of destruction of the right half of the sacrum.

The patient appeared to be well developed and only moderately ill, though he complained bitterly of pain in the right buttock. He was unable to stand and could barely maintain himself in a sitting position. He was most comfortable while lying on the left side with the right leg flexed. A bulging mass was present in the right gluteal region near the midline. It measured 12 cm. in diameter and was hard, non-pitting, and tender to light palpation. Rectal examination was difficult, due to extreme tenderness. No irregularity of the rectal mucosa was felt, although a large mass could be palpated posteriorly. The remaining positive findings were neurological in character and included atrophy of the right posterior thigh and gluteal region, absence of the right ankle jerk, motor weakness of the right leg, impairment of all types of sensation on the right over the areas of distribution of the 5th lumbar nerve and all the sacral nerves, with similar though less severe impairment on the left. There were also hypesthesia of the skin of the penis and scrotum on the right and mild weakness of the anal sphincter.

---

[1] From the Departments of Radiology, New York Medical College, Flower Fifth Avenue Hospitals, and the Veterans Administration Hospital, Lyons, N. J. Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the author.

Accepted for publication in July 1948.

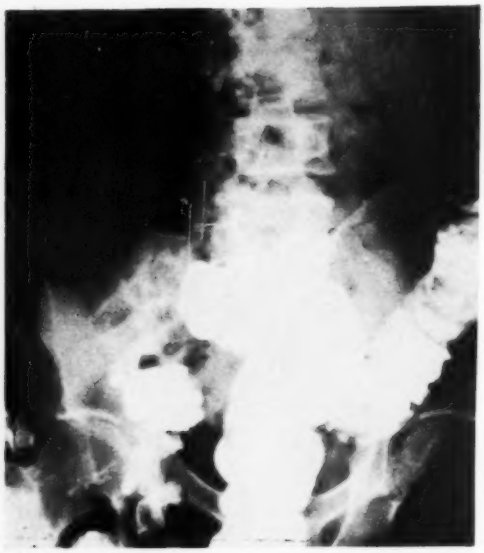


Fig. 2. Anteroposterior view of the pelvis, showing the relationship between the barium-filled rectum and the iodoform gauze-packed tumor cavity.

Impression: Cauda equina syndrome, etiology unknown.

Laboratory Data: Blood counts, urinalysis, and serologic tests were normal.

Roentgen examination of the pelvis (Fig. 1) revealed an irregular area of destruction of the greater portion of the right half of the sacrum, with ballooning out of a thin shell of bone. A large soft-tissue mass was demonstrated, extending from the osteolytic lesion.

Clinical Course: A biopsy was performed on the buttock mass; approximately 100 c.c. of pus and necrotic tissue were evacuated and the cavity was packed with iodoform gauze. Microscopic study showed necrotic bone infiltrated by hyperplastic columnar epithelium in glandular arrangement. The pathologist suggested that the tumor was derived from the gastro-intestinal tract.

A barium enema study showed marked anterior displacement of the rectum by an ill-defined soft-tissue mass (Figs. 2 and 3). There was no irregularity of the posterior rectal wall and no communication was demonstrated between the rectum and the gauze-filled tumor cavity. The remainder of the colon was negative.

Roentgenograms of the chest, extremities, and lumbar spine were negative. Fluoroscopic study of the stomach with a small amount of barium showed no abnormalities.

The patient ran a downhill course. A colostomy was performed prior to palliative roentgen therapy, but death occurred shortly thereafter of peritonitis.

Postmortem Findings: At autopsy, a flat tumor was found on the posterior wall of the rectum, about 3 cm. proximal to the sphincter. The tumor measured 3 cm. in diameter and had scalloped margins and a firm granular base. There was no extension into the perirectal tissues. The presacral, sacral, and buttock area on the right was occupied by a firm gritty mass, measuring 9 cm. in diameter and easily separated from the perirectal tissues. Numerous small metastatic nodules were present in the liver. No other significant findings were observed.

Fig. 3. Lateral view (retouched) of the pelvis, showing anterior displacement of the rectum by the tumor.

Microscopic study showed numerous nests of columnar epithelium supported by scant stroma. The epithelial cells were arranged in glandular formation. No tumor continuity was demonstrated between the rectal lesion and the sacral mass.

DISCUSSION

The cauda equina syndrome results from involvement of the nerve roots of the lumbar and sacral segments. The syndrome may be the result of trauma, inflammation, or tumor. Among the manifestations are root pain, limb weakness progressing to flaccid paralysis, and impairment of all forms of sensation in the affected roots (4). The levels and regions affected depend on the particular roots involved. In the presence of low lesions, the bladder and rectum are affected, with sensory changes in the form of saddle anesthesia. Sex urge may be abolished.

In seeking the cause of a cauda equina syndrome, roentgenographic studies must be considered essential. Digital and endoscopic evaluation of tumors displacing or involving the rectum is frequently difficult. Roentgenograms of the pelvis and lumbosacral region may show an area of increased density or osseous destruction which would point toward tumor rather than some other cause of the symptoms. Regardless of the findings, barium enema studies should be performed, with particular emphasis on oblique and lateral views. Such views may not only disclose filling irregularities of the rectal wall not seen on the anteroposterior views, but they also will allow evaluation of the presacral space. Ordinarily the rectum follows closely the contours of the sacrum, and even small deviations in the width of the space may be significant in providing a clue to a lesion in this region.

Among the tumors encountered are dermoid, chordoma, ependymoma, teratoma, endothelial myeloma, giant-cell tumor, neurofibroma, metastatic carcinoma, lymphoblastoma, and myeloma (5). The type of tumor can be determined only by histologic study.

SUMMARY

A case of cauda equina syndrome with an underlying "silent" rectal carcinoma is presented.

The importance of roentgenographic examination with proper views for determining the presence of a tumor in such cases is stressed.

Knollcroft Road
Lyons. N. J.

REFERENCES

1. KIRSNER, J. B., AND PALMER, W. L.: Errors in Diagnosis of Neoplastic Lesions of the Rectum, Rectosigmoid, and Colon. M. Clin. North America **28:** 278–286, January 1944.
2. MAYO, C. W., AND SCHLICKE, C. P.: Carcinoma of Colon and Rectum: Study of Metastasis and Recurrences. Surg., Gynec. & Obst. **74:** 83–91, January 1942.
3. ACKERMAN, L. V., AND DEL REGATO, J. A.: Cancer: Diagnosis, Treatment, and Prognosis. St. Louis, Mo., C. V. Mosby Co., 1947.
4. WECHSLER, I. S.: Textbook of Clinical Neurology. Philadelphia, W. B. Saunders Co., 5th ed., 1943.
5. BRINDLEY, G. V.: Sacral and Presacral Tumors. Ann. Surg. **121:** 721–736, May 1945.

SUMARIO

Síndrome de la Cola de Caballo, Debido a Carcinoma Rectal Silencioso

El caso descrito de síndrome de la cola de caballo se debía a subyacente carcinoma rectal "silencioso" con metástasis en el sacro.

Al tratar de determinar la causa de un síndrome de la cola de caballo, los estudios radiográficos revisten importancia si los estudios digitales y endoscópicos resultan imprecisos. Las radiografías de la pelvis y la región sacrolumbar pueden revelar una zona de mayor densidad o de osteólisis que denote tumor más bien que alguna otra causa. Independientemente de los hallazgos, deben ejecutarse estudios con enemas de bario, haciendo hincapié en las vistas oblicuas y laterales. Esas vistas pueden no tan sólo revelar nichos de la pared rectal que no aparecen en las anteroposteriores, sino también permitir la valuación del espacio presacro. Hasta las más pequeñas modificaciones en el tamaño de dicho espacio pueden resultar importantes en lo tocante a facilitar algún indicio de la enfermedad presente en dicha región.

EDITORIAL

The Role of the Radiologist in Mass Chest X-Ray Survey

Tuberculosis, the leading cause of death in the age group fifteen to forty-five, still presents a challenge to the medical profession. It is the only one of nine leading causes of death that we possess the knowledge to eradicate. Since every case is acquired from some infected person, the public health problem is to find those who have the disease and provide sufficient isolation to prevent further transmission. Until the advent of mass chest x-ray surveys, various methods of case finding were employed. The chief reliance was placed on a follow-up of contacts in cases of known disease. This remains an important procedure and is a necessary corollary of the mass survey method.

In addition to case finding, a community-wide survey provides a base line for statistical proof of disease incidence, and thus an accurate measure for the needs of a community for public health staff, sanitariums, and hospital accommodations. A repetition of the survey after a definite time interval would provide reasonably accurate knowledge as to the proficiency of preventive measures and an approximation of the attack rate.

It is interesting to note the practicability of a community-wide chest survey program hinged on the demonstration that efficient chest film readings can be accomplished in fully clothed patients. The conclusions formed from the original investigative work on this simplified procedure have been amply proved in the large surveys already undertaken.

The approval of organized medicine in a community is a basic requirement before the United States Public Health Service will lend its aid to mass chest survey projects. The physicians in each of the communities so far covered have had important duties. They have aided in the organization of professional and technical phases of the campaign, correlating the work of the physician, public health authorities, and statisticians. They have guided the lay staff in planning a system of case reading, notification of findings, and follow-up management. They have lent assistance in guiding publicity so that citizens are not terrorized by over-enthusiastic scare propaganda, nor lulled into the security that a chest film is a substitute for a complete physical check-up.

Radiologists working on these professional committees have also helped carry on educational work among the physicians. This has taken the form of a "refresher course" type of meeting and bulletins designed to instruct physicians in follow-up diagnostic procedures, case handling, availability of local nurse and social worker aid, the use and limitations of antibiotics, and other timely topics. The radiologists of the community have the further responsibility of reassuring the citizens and the medical profession that the film reading is being done on as high a plane of accuracy as is possible. Fortunately, this has presented no problem. The film readers are trained United States Public Health physicians, schooled under the exacting supervision of Dr. Ira Lewis. They work in teams of four to six under the supervision of trained radiologists. Cross-checking and informal consultation are the rule. This stimulating environment makes for a lively critical study of the 70-mm. films. There is no doubt that as training systems are improved through the analytical work now in progress (1, 2) further accuracy will be attainable.

In the case of patients returning for 14 × 17-inch film study, history and further

clinical data are available to evaluate chest findings. These larger films are studied by the Public Health film readers, and all doubtful cases are held for study by a review board. The review boards are organized on a volunteer basis from the radiologists and other physicians in the community interested in chest diseases. In Cleveland, Ohio, thirty-five radiologists and twenty chest specialists were formed into teams of two or three each, who rotated the duty of meeting twice a week to dispose of doubtful cases. In serving on these review boards, Cleveland radiologists have confirmed many findings of the expert investigators who have done comparison film study (2). There is a lack of standard terminology; such words as exudative, soft, fibrotic, honeycombed, veiled, nebulous, productive, spotted, and the like, are without any semblance of standard usage. The problem of semantics thus posed is in need of serious study. These review board sessions have also substantiated the inherent personal difference in "seeing" of any group of presumably qualified men. There have been no attempts to prove *inter-* or *intra*-individual variations of film reading, but there has been ample proof that these variations occur, as previous investigators have shown.

There have been hopes that the mass chest x-ray surveys would disclose a significant number of early carcinomas of the lungs. The follow-up study on such suspects has naturally been slow in all cities so far. There is little indication, however, that this will turn out to be an important by-product of the investigation, even though the film readers are alert to the possibility.

Thus the advent of mass chest x-ray surveys in a community imposes definite responsibilities on the radiologists, but there are genuine rewards for this added work. There is an opportunity to evaluate one's acumen and opinions against other radiologists, and to compare them with the opinions of chest physicians, whose approach to the problem of film diagnosis is somewhat different from our own. There is an opportunity to see a large number of widely differing anomalies, chest diseases, and their residua. Finally, an opportunity is afforded to participate in a worth-while health project with the expectation that present and future benefits may accrue to all the community.

GEORGE L. SACKETT, M.D.

REFERENCES

1. GARLAND, L. HENRY: On the Scientific Evaluation of Diagnostic Procedures. Radiology **52:** 309–328, March 1949.

2. BIRKELO, C. C., AND OTHERS: Tuberculosis Case Finding: A Comparison of Effectiveness of Various Roentgenographic and Photofluorographic Methods. J. A. M. A. **133:** 359–366, Feb. 8, 1947.

ANNOUNCEMENTS AND BOOK REVIEWS

ANNUAL MEETING RADIOLOGICAL SOCIETY OF NORTH AMERICA

The Thirty-fifth Annual Meeting of the Radiological Society of North America will be held in Cleveland, Ohio, Dec. 4 to 9, 1949, with headquarters at the Cleveland Public Auditorium and the Statler Hotel. Arrangements already in progress promise an outstanding meeting, with a notable program, carefully planned refresher courses, and extensive scientific and commercial exhibits with adequate space for effective displays. Further details as to the meeting will appear in RADIOLOGY for September and the following months.

ARIZONA ASSOCIATION OF PATHOLOGISTS AND RADIOLOGISTS

The Arizona Association of Pathologists and Radiologists has elected as its officers for the ensuing year Dr. Ralph Fuller of Tucson as President, Dr. James H. West, also of Tucson, as Vice-President, and Dr. R. Lee Foster, 507 Professional Building, Phoenix, as Secretary-Treasurer. Meetings of the new society will be held in connection with the annual meeting of the State Medical Association and six months thereafter, and at the call of the secretary.

FLORIDA RADIOLOGICAL SOCIETY

The newly elected officers of the Florida Radiological Society are: John A. Beale, M.D., of Jacksonville, President; F. K. Hurt, M. D., of Jacksonville, Vice-President; John J. McGuire, M.D., 1117 N. Palafox, Pensacola, Secretary-Treasurer.

The annual fall meeting of the Society will be held in October or early in November.

KINGS COUNTY RADIOLOGICAL SOCIETY

At a recent meeting of the Long Island Radiological Society (New York), the name of the organization was changed to Kings County Radiological Society. The new officers are: President, Dr. H. G. Koiransky; Vice-President, Dr. I. Silverstein; Treasurer, Dr. M. H. Goldenberg; Secretary, Dr. Marcus Wiener, 1430 48th St., Brooklyn 19, N. Y.

RADIOLOGICAL SOCIETY OF NEW JERSEY

The Radiological Society of New Jersey has elected the following officers for the ensuing year: President, F. B. Carrigan, M.D.; Vice-President, Raphael Pomeranz, M.D.; Secretary, Benjamin Copleman, M.D., 280 Hobart St., Perth Amboy, N. J.; Treasurer, C. A. Plume, M.D.; Counselor, R.S.N.A., P. J. Gianquinto, M.D., and Counselor, A.C.R., W. H. Seward, M.D.

NEW YORK ROENTGEN SOCIETY

The officers of the New York Roentgen Society recently elected to serve for the year Oct. 1, 1949-Oct. 1, 1950 are: President, Dr. William Snow; Vice-President, Dr. Robert P. Ball; Secretary, Dr. F. H. Ghiselin; Treasurer, Dr. Harold L. Temple.

THIRD INTER-AMERICAN CONGRESS OF RADIOLOGY

Plans for the Third Inter-American Congress of Radiology to be held in Santiago, Chile, Nov. 11–17, 1949, are rapidly approaching completion. The Government of Chile has officially recognized the Congress and has lent the official patronage of the University of Chile, which means that it has arranged for the assistance of the Faculty of Medicine in connection with the Congress and through it has invited eminent radiologists and cancerologists from other countries to participate.

The meeting will be held in the Hotel Crillon, situated in the very commercial and social center of Santiago, a short distance from the Government Palace, the University of Chile, and from the other principal hotels.

The official program for the meeting, as previously announced (see RADIOLOGY for April 1949) will be devoted to (A) Radiological Exploration of the Cardiovascular System with Opaque Material, (B) Diagnosis and Simple Radiological Exploration of the Skull, (C) Radiation Treatment of Cancer of the Tongue, and (D) Radiation Treatment of Cancer of the Cervix.

The United States has been assigned an official essayist and a collaborator for each of the four official theses, and these may present one or more co-lecturers, as convenient.

Papers which do not have a direct relation to the official themes may be presented in the scientific exhibit, time apart from the formal sessions having been set aside for this purpose.

There will be bilingual presentations of the Proceedings of the meeting, RCA Chilena having charge of the installation necessary for the immediate translation and transmission of the official languages of the Congress.

Special itineraries have been prepared for the journey, including travel by air, land, and sea. The municipality of Viña del Mar, one of the most attractive watering places in South America, proposes to invite those in attendance at the Congress to participate in a two-day program in that city, and other sightseeing tours will undoubtedly be offered for our pleasure. For those who have definitely decided to attend the meeting, catalogues describing the beauties of southern Chile are available.

Data regarding passports and other essential

The Cleveland Public Auditorium, where the sessions of the Annual Meeting of the Radiological Society of North America will be held and the Scientific and Commercial Exhibits will be shown.

documents which must be carried can be obtained from the undersigned. For those who are to participate in the program, the committee has special instructions regarding the preparation of manuscripts, illustrations, charts, tables, etc.

All those who plan to attend, no matter under whose auspices or by what travel plan, should communicate with the undersigned, sending twenty dollars to insure enrollment. Membership does not depend on attendance at the Congress. One can be a member and receive a copy of the Proceedings without attending.

JAMES T. CASE, M.D.
Regional Secretary
55 East Washington St.,
Chicago 2, Ill.

In Memoriam

PERCY JOSEPH DELANO, M.D.

Dr. Percy Joseph Delano, of Chicago, met an accidental death on Jan. 14, 1949. Dr. Delano was born in Kewanee, Ill., in 1899. He was graduated in medicine from the University of Illinois in 1926, served his internship in Cook County Hospital from 1926 to 1928, and entered practice in Chicago, serving as Instructor in Surgery at his *alma mater* from 1928 to 1937. During the years 1937 and 1938 Dr. Delano was a Fellow in Radiology at Cook County Hospital under the direction of the late Dr. Maximilian J. Hubeny. He was a diplomate of the American Board of Radiology, a member of the Chicago Medical Society, the Illinois State Medical Society, the Radiological Society of North America, the American Roentgen Ray Society, and the American College of Radiology, and a Fellow of the American Medical Association and of the Chicago Roentgen Society. For a number of years he was Radiologist to West Suburban Hospital, Oak Park, Ill.

Dr. Delano was very active in the affairs of the Chicago Roentgen Society, contributing frequent case reports to the scientific meetings. He twice received the award for the outstanding report of the year. He served long and faithfully as an abstractor for RADIOLOGY.

Dr. Delano's interest in all that concerned radiology was unfailing, and his contributions to the specialty will be greatly missed by his colleagues, particularly in Chicago.

Dr. Delano was married in 1929 to Miss Lucy Hartman, who survives him.

WARREN W. FUREY, M.D.

WALTER C. POPP, M.D.

Dr. Walter C. Popp was born in Pittsburgh, Penna., Oct. 24, 1901. He was graduated from St. Vincent College, Latrobe, Penna., in 1925 and received his degree of doctor of medicine from the University of Pittsburgh in 1929. After serving his internship at St. Francis Hospital, Pittsburgh, he entered the Mayo Foundation as a Fellow in Dermatology and Syphilology in 1930. In 1933 he received his master of science degree from the University of Minnesota with a thesis on the subject of "Reaction of Skin to Grenz Rays."

Walter C. Popp, M.D.

Dr. Popp became interested in roentgen therapy of dermatologic and systemic disease conditions while working in the department of dermatology, and in 1934 was made an associate in roentgen therapy on the staff of the Mayo Clinic. He was appointed instructor in roentgenology on the Mayo Foundation in 1935. He was a member of the American Medical Association and the Radiological Society of North America. At the time of his death on June 4, 1949, he was a consultant in therapeutic radiology in the Mayo Clinic and Assistant Professor of Roentgenology in the Mayo Foundation. Death came suddenly from coronary occlusion.

During the last five years of his life, Dr. Popp made a singular contribution by his management of regional ileitis with roentgen therapy. Up to the time of his death he had treated fifty patients, some with very striking results.

Dr. Popp was extremely active in community civic affairs. He was President of the Rochester Automobile Club, President of the City-County Safety Council, and President of the Lourdes Parent-Teachers Association. He gave much time to the work of the Chamber of Commerce and served on the committees on conventions, highways, and traffic safety. The profession and community have suffered a grievous loss.

It was the good fortune of this writer to work with Dr. Popp in rather close association on some problems. He embodied all that is noble and fine in a physician. He gave unstintingly to those under his care and showed gentleness and patience at all times. Association with him, both professionally and socially, was always a stimulation.

His home life, too, was ideal, and his love for a devoted wife and his three daughters, all of whom survive him, was unfailing. He and Mrs. Popp generously extended their hospitality to a large circle of friends, and many memorable evenings were spent at their home. Life was made richer for many for having known and been associated with Dr. Walter C. Popp, and his memory will be with us always.

J. A. BARGEN, M.D.

Books Received

Books received are acknowledged under this heading, and such notice may be regarded as recognition of the courtesy of the sender. Reviews will be published in the interest of our readers and as space permits.

INTRODUCTION TO RADIOCHEMISTRY. By GERHART FRIEDLANDER, Chemist, Brookhaven National Laboratory (Visiting Lecturer, Washington University, St. Louis), and JOSEPH W. KENNEDY, Professor of Chemistry, Washington University, St. Louis. A volume of 412 pages. Published by John Wiley & Sons, Inc., 440 Fourth Ave., New York 16, N. Y., 1949. Price $5.00.

RADIOACTIVE TRACER TECHNIQUES. By GEO. K. SCHWEITZER, Assistant Professor of Chemistry, University of Tennessee, and IRA B. WHITNEY, Chief Supervisor, Radio Chemical Process Development, Oak Ridge National Laboratories. A volume of 242 pages, with 13 illustrations. Published by D. Van Nostrand Co., New York, 1949. Price $3.25.

REGIONAL ILEITIS. By BURRILL B. CROHN, M.D., Consulting Gastroenterologist, Mount Sinai Hospital, New York. A volume of 230 pages, with 74 illustrations. Published by Grune & Stratton, New York, 1949. Price $5.50.

INVESTIGATIONS INTO DIFFERENTIATION AND OTHER MORPHOLOGICAL CHANGES IN MALIGNANT TUMORS FOLLOWING THERAPEUTIC IRRADIATION WITH X-RAYS AND RADIUM. By S. RY ANDERSEN. Treatise for the Doctorate at the University of Copenhagen. A volume of 112 pages, with 28 photomicrographs. Published by Einar Munksgaard, Copenhagen, 1949.

EL NEUMOMEDIASTINO ANTERIOR ARTIFICIAL EN EL NIÑO. SU IMPORTANCIA PARA EL ESTUDIO DE LA HIPERPLASIA TÍMICA. By ANDRÉS P. H.

DEGOY, Médico de la Casa Cuna, Córdoba, and SABINO DI RIENZO, Professor Adjunto de Radiología y Fisioterapia, Córdoba. A volume of 104 pages, with 56 illustrations. Published by Librería y Editorial "El Ateneo," Florida, 340, Córdoba 2099, Buenos Aires, Argentina, 1948.

APERÇUS ROENTGENTHÉRAPIQUES RELATIFS À DIVERS MODES D'INVOLUTION CANCÉREUSE, ET MÉTHODES DE PROTECTION. By Dr. HENRI COUTARD, with the collaboration of Dr. MACMULLEN, Director of the Penrose Tumor Clinic, Colorado Springs, Colo. A volume of 226 pages, with 6 illustrations. Published by G. Doin & Cie, Paris, 1949. Price 1, 200 fr.

RADIOLOGIA AUSTRIACA. Herausgegeben von der Österreichischen Röntgen-Gesellschaft. Band I. A volume of 130 pages, with 82 illustrations. Published by Urban & Schwarzenberg, Wien, 1948. Price $4.70. (Selected papers from this volume will be abstracted.)

Book Reviews

ROENTGEN DIAGNOSIS OF THE EXTREMITIES AND SPINE. Annals of Roentgenology, Vol. 17. By ALBERT B. FERGUSON, M.D., Associate Professor, Orthopaedic and Fracture Surgery, Boston University; Consulting Roentgenologist, Children's and Memorial Hospitals, Boston; Formerly Director of Roentgenology, New York Orthopaedic Hospital. A volume of 520 pages, with 625 roentgen-ray studies and 8 line cuts. Published by Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 2d ed., 1949. Price $15.00.

Dr. Albert B. Ferguson has revised and enlarged his work on Roentgen Diagnosis of the Extremities and Spine, first published in 1939 as Volume 17 of the Annals of Roentgenology. Two chapters on bone tumors with some seventy new illustrations have been added.

The general plan is not to consider exhaustively the various disease entities involving the extremities and spine, but rather to describe the bone as it appears in health and disease. In certain specific conditions, however, a fairly complete account of the roentgen changes is given, together with points of differential diagnosis.

In describing the pathology as revealed by the roentgenogram, the author has frequent recourse to physiology and the basic underlying associated changes. In certain instances his terminology is not that in common usage and reference to his definition of the terms is necessary. This may be confusing to the casual reader.

The text is amply illustrated with 631 figures with adequate captions and brief case histories. The illustrations are the less desirable positives rather than the negatives which would actually be seen by the roentgenologist, but in most instances are of good quality.

This book contains a great deal of helpful information for the discerning reader. It will be a useful addition to the library of the radiologist and orthopedist.

BIOLOGICAL REACTIONS CAUSED BY ELECTRIC CURRENTS AND BY X-RAYS. A THEORETICAL STUDY OF THE PHENOMENA OF EXCITATION IN THE NERVE BY DIFFERENT ELECTRIC CURRENTS AND OF THE BIOLOGICAL REACTIONS CAUSED BY X-RAYS, BOTH BASED UPON A COMMON PRINCIPLE. By J. TH. VAN DER WERFF, M.D., D.Sc., Radiologist of the St. Canisius Hospital, Nymegen, Netherlands. A volume of 204 pages, with 38 illustrations. Published by the Elsevier Publishing Co., New York, Amsterdam, London, Brussels, 1948. Price $5.00.

In his study of the biological reactions to electric currents and x-rays, Dr. van der Werff points out that for the last twenty-five years mathematical physical methods have been used to solve problems of purely biological character. The results achieved, he feels, have made biology too much like physics. His approach to these problems is from the biological point of view but he has made extensive use of mathematical physical methods in his explanations.

The book is divided into three parts. In the first part it is shown that the two subjects discussed, namely, the phenomena of excitation caused by electric currents, especially those of excitation of the nerve, and the biological reactions caused by x-rays, are analogous to such an extent that it is highly probable that they may be explained on the basis of a common principle. An attempt has been made to formulate this principle in order to apply it to the subjects under consideration. In the second part, the phenomena of local excitation of nerves are treated extensively. The third part deals with various radiological problems, including those of radiotherapy. Both the second and third parts end with a chapter called "Summary and Conclusions" in which mathematical formulas, except for the proposed equation, are omitted, so that a relatively simple explanation of the new theory may be presented to the reader.

RADIOLOGICAL SOCIETIES: SECRETARIES AND MEETING DATES

Editor's Note: Secretaries of state and local radiological societies are requested to co-operate in keeping this section up-to-date by notifying the editor promptly of changes in officers and meeting dates.

RADIOLOGICAL SOCIETY OF NORTH AMERICA. *Secretary-Treasurer*, Donald S. Childs, M.D., 713 E. Genesee St., Syracuse 2, N. Y.

AMERICAN RADIUM SOCIETY. *Secretary*, Hugh F. Hare, M.D., 605 Commonwealth Ave., Boston 15, Mass.

AMERICAN ROENTGEN RAY SOCIETY. *Secretary*, Harold Dabney Kerr, M.D., Iowa City, Iowa.

AMERICAN COLLEGE OF RADIOLOGY. *Secretary*, William C. Stronach, 20 N. Wacker Dr., Chicago 6, Ill.

SECTION ON RADIOLOGY, A. M. A. *Secretary*, U. V. Portmann, M.D., Cleveland Clinic, Cleveland 6, Ohio.

Alabama

ALABAMA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, W. D. Anderson, M.D., 420 10th St., Tuscaloosa.

Arizona

ARIZONA ASSOCIATION OF PATHOLOGISTS AND RADIOLOGISTS. *Secretary*, R. Lee Foster, M. D., 507 Professional Bldg., Phoenix.

Arkansas

ARKANSAS RADIOLOGICAL SOCIETY. *Secretary*, Fred Hames, M.D., Pine Bluff. Meets every three months and at meeting of State Medical Society.

California

CALIFORNIA MEDICAL ASSOCIATION, SECTION ON RADIOLOGY. *Secretary*, Sydney F. Thomas, M.D., Palo Alto Clinic, Palo Alto.

EAST BAY ROENTGEN SOCIETY. *Secretary*, Dan Tucker, 434 30th St., Oakland 9. Meets monthly. first Thursday, at Peralta Hospital.

LOS ANGELES RADIOLOGICAL SOCIETY. *Secretary*, Wybren Hiemstra, 1414 S. Hope St. Meets monthly, second Wednesday, County Society Bldg.

NORTHERN CALIFORNIA RADIOLOGICAL CLUB. *Secretary*, Charles E. Grayson, M.D., Medico-Dental Bldg., Sacramento 14. Meets at dinner last Monday of September, November, January, March, and May.

PACIFIC ROENTGEN SOCIETY. *Secretary*, L. Henry Garland, M.D., 450 Sutter St., San Francisco 8. Meets annually with State Medical Association.

SAN DIEGO ROENTGEN SOCIETY. *Secretary*, R. F. Niehaus, M.D., 1831 Fourth Ave., San Diego. Meets first Wednesday of each month.

X-RAY STUDY CLUB OF SAN FRANCISCO. *Secretary*, Wm F. Reynolds, M.D., University Hospital, San Francisco 22. Meets third Thursday at 7:45, January to June at Stanford University Hospital, July to December at San Francisco Hospital.

Colorado

COLORADO RADIOLOGICAL SOCIETY. *Secretary*, Mark S. Donovan, M.D., 306 Majestic Bldg., Denver 2. Meets third Friday of each month, at the Colorado School of Medicine and Hospitals.

Connecticut

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY. *Secretary*, Fred Zaff, M.D., 135 Whitney Ave., New Haven. Meetings bimonthly, second Wednesday.

CONNECTICUT VALLEY RADIOLOGICAL SOCIETY. *Secretary*, Ellwood W. Godfrey, M.D., 1676 Boulevard, W. Hartford. Meets second Friday of October and April.

District of Columbia

RADIOLOGICAL SECTION, DISTRICT OF COLUMBIA MEDICAL SOCIETY. *Secretary*, Alfred A. J. Den, M.D., 1801 K St., N.W., Washington 6. Meets third Thursday, January, March, May, and October, at 8:00 P.M., in Medical Society Auditorium.

Florida

FLORIDA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John J. McGuire, M.D., 1117 N. Palafox, Pensacola. Meets in April and in November.

Georgia

ATLANTA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Wm. W. Bryan, M.D., 490 Peachtree St., N. E. Meets second Friday, September to May.

GEORGIA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Robert Drane, M.D., De Renne Apartments, Savannah. Meets in November and at the annual meeting of State Medical Association.

Illinois

CHICAGO ROENTGEN SOCIETY. *Secretary*, John H. Gilmore, M. D., 720 N. Michigan Ave., Chicago 11. Meets at the Palmer House, second Thursday of October, November, January, February, March, and April at 8:00 P.M.

ILLINOIS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, William DeHollander, M.D., St. Johns' Hospital Springfield. Meetings quarterly as announced.

ILLINOIS STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY. *Secretary*, Harold L. Shinall, M.D., St. Joseph's Hospital, Bloomington.

Indiana

INDIANA ROENTGEN SOCIETY. *Secretary-Treasurer*, William M. Loehr, M.D., 712 Hume-Mansur Bldg., Indianapolis 4. Annual meeting in May.

Iowa

IOWA X-RAY CLUB. *Secretary*, Arthur W. Erskine, M.D., 326 Higley Building, Cedar Rapids. Meets during annual session of State Medical Society.

Kansas

KANSAS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Anthony F. Rossitto, M.D., Wichita Hospital, Wichita. Meets annually with State Medical Society.

Kentucky

KENTUCKY RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Everett L. Pirkey, M.D., 323 East Chestnut St., Louisville 2.

LOUISVILLE RADIOLOGICAL SOCIETY, *Secretary-Treasurer*, Everett L. Pirkey, Louisville General Hospital, Louisville 2. Meets second Friday of each month at Louisville General Hospital.

Louisiana

LOUISIANA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Johnson R. Anderson, M.D., No. Louisiana Sanitarium, Shreveport. Meets with State Medical Society.

ORLEANS PARISH RADIOLOGICAL SOCIETY. *Secretary*, Joseph V. Schlosser, M.D., Charity Hospital of Louisiana, New Orleans 13. Meets first Tuesday of each month.

SHREVEPORT RADIOLOGICAL CLUB. *Secretary*, Oscar O. Jones, M.D., 2622 Greenwood Road. Meets monthly September to May, third Wednesday.

Maryland

BALTIMORE CITY MEDICAL SOCIETY, RADIOLOGICAL SECTION. *Secretary*, J. Howard Franz, M.D., 1127 St. Paul St., Baltimore 2.

Michigan

DETROIT X-RAY AND RADIUM SOCIETY. *Secretary-Treasurer*, George Belanger, M.D., Harper Hospital, Detroit 1. Meetings first Thursday, October to May, at Wayne County Medical Society club rooms.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS. *Secretary-Treasurer*, R. B. MacDuff, M.D., 220 Genesee Bank Building, Flint 3.

Minnesota

MINNESOTA RADIOLOGICAL SOCIETY. *Secretary*, C. N. Borman, M.D., 802 Medical Arts Bldg., Minneapolis 2. Meets in Spring and Fall.

Missouri

RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY. *Secretary*, Wm. M. Kitchen, M.D., 1010 Rialto Building, Kansas City 6, Mo. Meetings last Friday of each month.

ST. LOUIS SOCIETY OF RADIOLOGISTS. *Secretary*, Charles J. Nolan, M.D., 737 University Club Bldg. Meets on fourth Wednesday, October to May.

Nebraska

NEBRASKA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Ralph C. Moore, M.D., Nebraska Methodist Hospital, Omaha 3. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England

NEW ENGLAND ROENTGEN RAY SOCIETY. *Secretary-Treasurer*, George Levene, M.D., Massachusetts Memorial Hospitals, Boston. Meets monthly on third Friday at Boston Medical Library.

New Hampshire

NEW HAMPSHIRE ROENTGEN SOCIETY. *Secretary*, Albert C. Johnston, M.D., Elliot Community Hospital, Keene. Meetings quarterly in Concord.

New Jersey

RADIOLOGICAL SOCIETY OF NEW JERSEY. *Secretary*, Benjamin Copleman, M.D., 230 Hobart St., Perth Amboy. Meetings at Atlantic City at time of State Medical Society and midwinter in Newark.

New York

ASSOCIATED RADIOLOGISTS OF NEW YORK, INC. *Secretary*, William J. Francis, M.D., East Rockaway.

BROOKLYN ROENTGEN RAY SOCIETY. *Secretary*, J. Daversa, M.D., 603 Fourth Ave., Brooklyn. Meets fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY. *Secretary-Treasurer*. Mario C. Gian, M.D., 610 Niagara St., Buffalo 1. Meetings second Monday, October to May.

CENTRAL NEW YORK ROENTGEN SOCIETY. *Secretary*, Dwight V. Needham, M.D., 608 E. Genesee St., Syracuse 10. Meetings January, May, October.

KINGS COUNTY RADIOLOGICAL SOCIETY. *Secretary*, Marcus Wiener, M.D., 1430 48th St., Brooklyn 19. Meetings fourth Thursday evening, October to May, at 8:45 P.M., in Kings County Medical Bldg.

NEW YORK ROENTGEN SOCIETY. *Secretary*, F. H. Ghiselin, M.D., 111 E. 76 St., New York.

QUEENS ROENTGEN RAY SOCIETY. *Secretary*, Jacob E. Goldstein, M.D., 88-29 163rd St., Jamaica 3. Meets fourth Monday of each month.

ROCHESTER ROENTGEN-RAY SOCIETY. *Secretary-Treasurer*, Ralph E. Alexander, M.D., 101 Medical Arts Bldg., Rochester 7. Meets at Strong Memorial Hospital, third Monday, September through May.

North Carolina

RADIOLOGICAL SOCIETY OF NORTH CAROLINA. *Secretary*, James E. Hemphill, M.D., Professional Bldg., Charlotte 2. Meets in May and October.

North Dakota

NORTH DAKOTA RADIOLOGICAL SOCIETY. *Secretary*, Charles Heilman, M.D., 1338 Second St., N., Fargo.

Ohio

OHIO STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Carroll Dundon, M.D., 2065 Adelbert Road, Cleveland 6. Meets with State Medical Association.

CENTRAL OHIO RADIOLOGICAL SOCIETY. *Secretary*, Paul D. Meyer, M.D., Grant Hospital, Columbus. Meets second Thursday, October, December, February, April, and June, 6:30 P.M., Seneca Hotel, Columbus.

CINCINNATI RADIOLOGICAL SOCIETY. *Secretary*, Eugene L. Saenger, M.D., 735 Doctors Bldg., Cincinnati 2. Meets last Monday, September to May.

CLEVELAND RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John R. Hannan, M.D., Cleveland Clinic, Cleveland 6. Meetings at 6:30 P.M. on fourth Monday, October to April, inclusive.

Oklahoma

OKLAHOMA STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, W. E. Brown, M.D., 21st and Xanthus, Tulsa 4. Meets in October, January, and May.

Oregon

OREGON RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Boyd Isenhart, M.D., 214 Medical-Dental Bldg., Portland 5. Meets monthly, on the second Wednesday, at 8:00 P.M., in the library of the University of Oregon Medical School.

Pacific Northwest

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Sydney J. Hawley, M.D., 1320 Madison St., Seattle 4, Wash. Meets annually in May.

Pennsylvania

PENNSYLVANIA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, James M. Converse, M.D., 416 Pine St., Williamsport 8. Meets annually.

PHILADELPHIA ROENTGEN RAY SOCIETY. *Secretary*, Arthur Finkelstein, M.D., Graduate Hospital, Philadelphia. Meets first Thursday of each month at 8:00 P.M., from October to May, in Thomson Hall, College of Physicians, 21 S. 22d St.

PITTSBURGH ROENTGEN SOCIETY. *Secretary-Treasurer*, R. P. Meader, M.D., 4002 Jenkins Arcade, Pittsburgh 22. Meets second Wednesday of each month at 6:30 P.M., October to June.

Rocky Mountain States

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Maurice D. Frazer, M.D., Lincoln Clinic, Lincoln, Nebr. Next meeting in Denver, Colo., Aug. 18–20, 1949.

South Carolina

SOUTH CAROLINA X-RAY SOCIETY. *Secretary-Treasurer*, Robert B. Taft, M.D., 103 Rutledge Ave., Charleston 16.

South Dakota

RADIOLOGICAL SOCIETY OF SOUTH DAKOTA. *Secretary-Treasurer* Marianne Wallis, M.D., 1200 E. Fifth Ave., Mitchell. Meets during Annual Session of State Medical Society.

Tennessee

MEMPHIS ROENTGEN CLUB. Meetings second Tuesday of each month at University Center.

TENNESSEE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, J. Marsh Frére, M.D., 707 Walnut St., Chattanooga. Meets annually with State Medical Society in April.

Texas

DALLAS-FORT WORTH ROENTGEN STUDY CLUB. *Secretary*, X. R. Hyde, M.D., Medical Arts Bldg., Fort Worth 2. Meetings on third Monday of each month in Dallas in the odd months and in Fort Worth in the even months.

HOUSTON X-RAY CLUB. *Secretary*, Curtis H. Burge, M.D., 3020 San Jacinto, Houston 4. Meetings fourth Monday of each month.

TEXAS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, R. P. O'Bannon, M.D., 650 Fifth Ave., Fort Worth. Next meeting Feb. 3–4, 1950, in Dallas.

Utah

UTAH STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Angus K. Wilson, M.D., 343 S. Main St., Salt Lake City. Meets third Wednesday, January, March, May, September, November.

Virginia

VIRGINIA RADIOLOGICAL SOCIETY. *Secretary*, P. B. Parsons, M.D., Norfolk General Hospital, Norfolk 7.

Washington

WASHINGTON STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Homer V. Hartzell, M.D., 310 Stimson Bldg., Seattle 1. Meetings fourth Monday, October through May, at College Club, Seattle.

Wisconsin

MILWAUKEE ROENTGEN RAY SOCIETY. *Secretary-Treasurer*, Theodore J. Pfeffer, M.D., 839 N. Marshall St., Milwaukee 2. Meets monthly on second Monday at the University Club.

RADIOLOGICAL SECTION OF THE WISCONSIN STATE MEDICAL SOCIETY. *Secretary*, S. R. Beatty, M.D., 185 Hazel St., Oshkosh. Two-day meeting in May; one-day with State Medical Society, September.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE. Meets first and third Thursdays 4 P.M., September to May, Service Memorial Institute, Madison 6.

Puerto Rico

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA. *Secretary*, Jesús Rivera Otero, M.D., Box 3542, Santurce, Puerto Rico.

CANADA

CANADIAN ASSOCIATION OF RADIOLOGISTS. *Honorary Secretary-Treasurer*, E. M. Crawford, M.D. Associate Honorary Secretary-Treasurer, Jean Bouchard, M.D. *Central Office*, 1535 Sherbrooke St., West, Montreal 26, Quebec. Meetings in January and June.

LA SOCIÉTÉ CANADIENNE-FRANÇAISE D'ELECTROLOGIE ET DE RADIOLOGIE MÉDICALES. *General Secretary*, Origéne Dufresne, M.D., Institut du Radium, Montreal. Meets third Saturday each month.

CUBA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA. Offices in Hospital Mercedes, Havana. Meets monthly.

MEXICO

SOCIEDAD MEXICANA DE RADIOLOGÍA Y FISIOTERAPIA. *General Secretary*, Dr. Dionisio Pérez Conío, Marsella 11, México, D. F. Meetings first Monday of each month.

ABSTRACTS OF CURRENT LITERATURE

ROENTGEN DIAGNOSIS

The Head and Neck

The Chest

The Digestive System

The Spleen

The Musculoskeletal System

Gynecology and Obstetrics

The Genito-Urinary System

The Adrenals

Hydatid Disease

Technic

RADIOTHERAPY

RADIOACTIVE ISOTOPES

EFFECTS OF RADIATION

ROENTGEN DIAGNOSIS

THE HEAD AND NECK

Pneumoencephalography in the Diagnosis of Cerebellar Atrophies. Report of Five Cases L. Uzman. Am. J. Roentgenol. **60:** 293–302, September 1948.

Pneumoencephalography is suggested as a useful procedure for an *in vivo* confirmation of the diagnosis of primary cerebellar atrophy, demonstrating a reduction in the size of the cerebellum in relation to the posterior fossa.

The reduction in the size of the cerebellum is not always in proportion to the intensity of the symptoms, nor does the apparent size of the cisterna cerebellomedullaris, as seen on a lateral film, necessarily give a true indication of the real size of the cerebellum in relation to the posterior fossa. Only a systematic study of infratentorial air pockets in stereoscopic anteroposterior, postero-anterior, and lateral views will give a true picture of the size of these pockets, and, therefore, an indication of the actual loss in cerebellar mass. The clinical diagnosis may thus be confirmed by pneumoencephalography but never ruled out by it.

The author describes his technic and gives the histories of 5 cases. The symptomatology was similar, the most common complaint being difficulty in walking. Cerebellar asynergia, dysmetria, and tremor were present in all 5 cases, minimally in the arms but most conspicuously in the legs. There was a history of alcoholism in 2 patients; the other 3 were "more or less confirmed abstainers." None had syphilis. Pneumoencephalography showed enlargement of the cisterna cerebellomedullaris in all 5 cases. In 1 case the individual folia could be seen outlined by subarachnoid air, suggestive, in itself, of a diagnosis of cerebellar atrophy.

Eight roentgenograms. EUGENE R. KUTZ, M.D.
Baltimore (Md.) City Hospitals

Calcified Intracranial Tuberculosis. H. Stephen Weens. J. Pediat. **33:** 328–335, September 1948.

The author calls attention to the rarity of calcification in tuberculomas of the brain and to the increased interest in the surgical treatment since it has been found that a fatal meningitis is not an invariable sequel of intervention. Diagnostically, calcified tuberculomas are of importance since they present certain roentgenologic features which may permit their differentiation from other calcifying intracranial lesions. These are described by Weinberger and Grant (Am. J. Roentgenol. **47:** 525, 1942. Abst. in Radiology **40:** 102, 1943). The calcifications vary in size and number but are more frequently solitary. They usually have a homogeneous center but a serrated, lace-like, angular margin. In some lesions, small calcium spicules and plaques seem to lie separated from the main center of calcification. The tuberculomas may in this way lose their compact structure, becoming irregular in outline, and may vary greatly in their dimensions. Among conditions requiring differentiation are craniopharyngiomas, angiomas, subdural hematomas, and the calcifications of tuberous sclerosis and toxoplasmosis.

Clinical signs and symptoms of isolated calcified tuberculomas depend on the site of the lesion. In some cases a tuberculous meningitis may overshadow all else; in others, symptoms referable to cerebral involvement may be absent. All of the author's 3 cases were of this last type.

The first patient, a colored girl of twenty months, showed no cerebral manifestations up to the time of her terminal illness, though multiple areas of calcification were demonstrable roentgenologically and at autopsy. The second patient was a fifteen-month-old colored boy with both pulmonary and osseous tuberculosis and four serrated calcified intracranial lesions assumed to be tuberculomas in view of the characteristic roentgen picture and the findings in the lungs and bones. The third case was that of a white child of nine years with a large calcified lesion discovered incidentally and assumed but not proved to be a tuberculoma.

Six roentgenograms. EUGENE R. KUTZ, M.D.
Baltimore (Md.) City Hospitals

Calcifying Epileptogenic Lesions. Hemangioma Calcificans: Report of a Case. Wilder Penfield and Arthur Ward. Arch. Neurol. & Psychiat. **60:** 20–36, July 1948.

In a six-year period 12 peculiar calcified neoplasms (or malformations) in the temporal lobe associated with epilepsy were studied by the authors in the Montreal Neurological Institute. Five tumors were classified as hemangioma calcificans. Of the 7 remaining cases, 2 were racemose hemangiomas, 1 a bony arachnoidal plaque, 2 proved to be post-traumatic degenerative calcification of an intercerebral hematoma, and 2 were believed to be tuberculomas but were not verified.

The present paper is devoted to hemangioma calcificans. This is a non-specific term denoting an angioma, not of the racemose type, that is predisposed to peculiar degenerative calcification. Basically the lesion is composed of vessels resembling veins, interspersed capillaries, and occasional atypical arterioles. The vessels are usually thin-walled, though thickening due to hyaline transformation was sometimes observed. The thick-walled vessels are frequently thrombosed.

Positive roentgen diagnosis of hemangioma calcificans is usually not possible. Some of the authors' cases showed small granules scattered through the tumor. In 2 the calcifications appeared in circumscribed masses and the tumor resembled a calcified tuberculoma, but in neither case was the peripheral outline quite so irregular.

Twelve roentgenograms; 4 photomicrographs; 4 photographs. JOHN R. HANNAN, M.D.
Cleveland Clinic Foundation

Tumors of the Septum Pellucidum. John D. French and Paul C. Bucy. J. Neurosurg. **5:** 433–449, September 1948.

The clinical and pathological data on 5 cases of tumor of the septum pellucidum are presented. Three of these tumors (possibly 4) were subependymal astrocytomas and 1 was a cellular ependymoma. Two tumors were resected; 1 patient is alive and well eleven years after operation and the other for a much shorter time.

Thirty-one similar recorded cases are reviewed. Only 3 (possibly 4) were successfully operated upon, and these all within the last five years. Gliomos of various kinds were diagnosed microscopically in these cases; in 6 instances the published material strongly suggests that they were subependymal astrocytomas. The probable origin of these various tumors in the subependymal plate is discussed.

Tumors of the septum pellucidum chiefly affect young adults. The clinical manifestations of all space-occupying masses of the septum pellucidum are similar and are predominantly the result of local compression and obstruction. The common symptoms are: episodes characterized by headache, occasionally visual and aural disturbances, and less frequently cataplectic-like weakness; mental disturbances, particularly related to memory; occasionally convulsions; less commonly unsteadiness or weakness of movement or numbness. Predominant findings are mental defects, frequently papilledema, less commonly pareses, paresthesias, ataxia, and defects of movement or tonus.

Roentgenographic studies are frequently diagnostic. The tumors were occasionally visualized as circumscribed areas of increased density in the midline well above the sella turcica. Calcification of the neoplasm was present in 5 of 32 cases (where sufficient data were included for evaluation), of which 4 were probably subependymal astrocytomas. From such roentgen evidence, other calcified tumors of the midline, such as gliomas of the corpus callosum or meningiomas of the falx, were considered frequently in the diagnosis. Ventriculography, however, dispelled all doubts in every case, showing dilated, separated lateral ventricles with biconcave medial borders.

Two roentgenograms; 4 photomicrographs; 3 tables.

One Hundred Cases of Miliary and Meningeal Tuberculosis Treated with Streptomycin. Paul A. Bunn. Am. J. M. Sc. **216:** 286–315, September 1948.

Detailed information is given on 100 patients with acute disseminated miliary tuberculosis and tuberculous meningitis. Treatment was initiated in the thirteen months subsequent to April 1946, and all but one of the survivors had been followed to October 1947. Of the 25 patients with combined miliary and meningeal tuberculosis, 4 were alive at the time of the report. Of 43 with meningeal tuberculosis alone, 16 survived, and of 10 with acute miliary tuberculosis, in whom meningitis developed during or after treatment with streptomycin, 3 were living. Of 22 patients with generalized miliary tuberculosis, without meningeal involvement, 16 were alive. There were thus 40 patients alive between four and fourteen months after initiation of treatment.

Roentgenographic evidence of pulmonary dissemination in these patients commenced to clear after sixty days of treatment and frequently went on to complete disappearance.

Those patients having pulmonary dissemination of miliary tuberculosis proved most responsive to streptomycin, but the results in tuberculous meningitis are especially noteworthy, since no other agent has proved able to lower the mortality appreciably below 100 per cent.

In an addendum, the authors furnish a further follow-up of the 40 surviving patients. Eight months after this paper was submitted for publication 15 of the 40 had died. Three of these were in the group originally described as having miliary tuberculosis alone, but in all 3 meningitis had developed. Seven of those with meningitis had died, and 5 of those with combined miliary and meningeal tuberculosis. Fifteen patients remained free of tuberculosis or showed arrested disease.

Six roentgenograms; 9 tables.

S. F. THOMAS, M.D.
Palo Alto, Calif.

Aneurysm of Terminal Portion of Anterior Cerebral Artery. O. Sugar and M. Tinsley. Arch. Neurol. & Psychiat. **60:** 81–85, July 1948.

Cerebral angiography is especially valuable in the accurate localization of intracranial aneurysms that otherwise might be difficult to diagnose, more particularly aneurysms at some distance from the circle of Willis.

The authors present a case in which an aneurysm near the posterior extremity of the pericallosal branch of the anterior cerebral artery was identified on both diodrast and thorotrast angiograms. The aneurysm was not visible on exploration, but in view of the angiographic findings the vessel was occluded by clips. The patient improved clinically, and postoperative cerebral angiograms showed no filling of the anterior cerebral artery or the aneurysm.

Two arteriograms.

JOHN R. HANNAN, M.D.
Cleveland Clinic Foundation

A Metastatic Lesion Simulating an Intracranial Aneurysm. Everett F. Hurteau. J. Neurosurg. **5:** 493–495, September 1948.

A case is reported which was diagnosed clinically, neurologically, and roentgenologically as aneurysm of the left internal carotid artery within the cavernous sinus. On the basis of the literature and the course of this case, it was felt that arteriography was not indicated. The patient died approximately one month after ligation of the left common carotid artery and two weeks following ligation of the left internal carotid artery in the neck. Autopsy revealed an infiltrating adenocarcinoma of the uterus which had metastasized to the nasopharynx, presumably by way of the paravertebral veins. There were no other metastases. The lesion in the nasopharynx had extended through the supra-orbital fissure and body of the sphenoid bone to fill completely both cavernous sinuses.

This case is offered as evidence that one may be misled by clinical signs and symptoms no matter how typical of aneurysm they may seem.

Two photographs.

Gargoylism. II. Study of Pathologic Lesions and Clinical Review of Twelve Cases. Stuart Lindsay, William Anthony Reilly, Thelma J. Gotham, and Richard Skahen. Am. J. Dis. Child. **76:** 239–306, September 1948.

This is a continuation of an earlier paper on gargoylism in which the clinical observations in 18 cases were reviewed (Am. J. Dis. Child. **75:** 595, 1948. Abst. in Radiology **52:** 455, 1949). The authors now present the clinical and pathological observations at autopsy in 8 cases and the clinical and surgical pathological findings in 4 additional cases. Some of their conclusions are:

(1) The basic lesion is the intracellular and extracellular deposition and storage of a substance giving the histochemical reactions for glycogen. (2) Certain histochemical data suggest that the glycogen may be combined with protein. (3) Laboratory studies indicate that the glycogen storage process in gargoylism is not associated with demonstrable alteration in carbohydrate metabolism, as in true glycogenosis (von Gierke's disease). (4) Widespread involvement of most tissues, including the nervous, cardiovascular, reticulo-endothelial, endocrine, skeletal, and other systems, explains the protean clinical manifestations of the disease. (5)

The lesions of gargoylism are similar to or identical with some of those of the other macromolecular storage diseases of both endogenous and exogenous origin. (6) Further, more precise histochemical, and enzymatic studies on the material stored in the tissues are indicated. Combination of glycogen with a protein stored in the cytoplasm of the cells may explain the inconsistent histochemical staining reactions to date.

Twenty-three illustrations.

Röntgen Ray Examination of the Paranasal Sinuses with Particular Reference to the Frontal Sinuses. Sölve Welin. Brit. J. Radiol. **21:** 431–437, September 1948.

Five standard projections are used in examination of the nasal sinuses, and one special projection. In the first position the patient is erect, his nose and forehead against the cassette, which is angulated 30 degrees toward the forehead. The beam is directed horizontally. The second position is similar to the first except that the angulation is only 20 degrees and the patient's nose and chin are held against the cassette, with the mouth open. The third position is a direct lateral, with the patient erect. The other two are submental-vertical projections, one with the beam running directly through the anterior plane of the face and the other with the head tilted so that the mandible falls in front of the frontal sinuses. The sixth position, and one only occasionally used, is a direct lateral with the patient recumbent.

Evidence of pathological change must be looked for in as many different projections as possible. Varying degrees of sclerosis or thickness of the bone may simulate a pathological process in the postero-anterior projections, particularly in the frontal sinuses. These sources of error will be picked up in the lateral and submento-vertical projections. The special lateral recumbent projection is useful in the presence of a large amount of fluid and only a small amount of air.

Fifteen roentgenograms; 6 diagrams.

SYDNEY J. HAWLEY, M.D.
Seattle, Wash.

Knife Blade Traversing Maxillary Antrum and Remaining in Nose Six Years. A. J. Vadala and Kenneth Somers. Mil. Surgeon **103:** 207–210, September 1948.

The essentials of the case here recorded are covered in the title. The knife blade traversed the maxillary antrum, transfixed the inferior turbinate, and its point finally pierced the superficial layers of the nasopharynx. The patient was stabbed in 1941; symptoms (headache and postnasal discharge) developed a year later, but the presence of the foreign body was not discovered until 1946, when it was demonstrated roentgenographically. It was successfully removed a year later.

Post-Traumatic Granuloma of the Bony Orbit Simulating Tumour. G. Stuart Ramsey, H. Wyatt Laws, J. E. Pritchard, and Harold Elliott. Canad. M.A.J. **59:** 206–211, September 1948.

Two cases of "post-traumatic granuloma" of the bony orbit are presented. Radiologically the lesions described can simulate any osteolytic lesion involving the frontal bone. In the cases reported the original impression was metastatic malignant tumor.

From a pathologic point of view, bone trauma with or without fracture may result in hematoma formation within bone. The sterile constituents of the broken down blood may act as a foreign body which may lie dormant for a considerable period and be "activated" following a subsequent injury or may cause a slow, steady reaction over several years. The "reaction" results in a destructive, expansile tumor which is actually a chronic granuloma.

Seventeen illustrations, including 4 roentgenograms.

JOHN DECARLO, JR., M.D.
Jefferson Medical College

Compression of the Cervical Spinal Cord by Herniated Intervertebral Discs. Paul C. Bucy, Robert F. Heimburger, and Harold R. Oberhill. J. Neurosurg. **5:** 471–492, September 1948.

Four cases of compression of the spinal cord by median herniations of cervical intervertebral disks are presented. The confusion of such cases with degenerative diseases of the spinal cord, such as multiple sclerosis, primary lateral sclerosis, and amyotrophic lateral sclerosis, and the differential diagnosis are discussed.

Median herniations of cervical intervertebral disks are characterized predominantly by spasticity and hyperreflexia in the lower extremities and by unsteadiness of gait. Sensory changes are commonly mild or absent. Pain, tenderness, and stiffness of the neck are uncommon. There may be paresthesias and muscular weakness and awkwardness in the upper extremities. Evidence of obstruction of the spinal canal (Queckenstedt's sign) is not present in many cases. The spinal fluid is normal except for a moderate elevation of the protein content in some cases. Myelography with lipiodol or pantopaque will reveal an obstruction or deformity in a majority of cases, but not all.

These herniations are best removed transdurally through a bilateral laminectomy of at least two vertebrae. The outcome of such operations in early cases should be excellent, but in severe cases of long duration poor results have not uncommonly been reported.

Nine roentgenograms; 1 drawing; 4 photomicrographs.

THE CHEST

An Analysis of Variations in the Bronchovascular Pattern of the Right Upper Lobe of Fifty Lungs. Edward A. Boyden and J. Gordon Scannell. Am. J. Anat. **82:** 27–74, January 1948.

The authors describe the prevailing mode of branching of the upper lobe bronchus as well as its most striking variations, and the prevailing pattern of the arteries and veins, as determined in complete dissections of 50 lobes. They conclude that the gross structure of the right upper lobe is extremely variable. To comprehend it, one must know not only the prevailing bronchial and vascular patterns but also the trends of their variation.

Eight color plates; 4 tables.

Case of Unilateral Pulmonary Agenesis with Ipsilateral Absence of the Diaphragm. Gregers Thomsen. Acta radiol. **30:** 191–196, September 1948.

Only fifty or sixty cases of pulmonary agenesis have been reported in the literature, most of which were seen at autopsy. The left lung is absent twice as often as the right. Death usually occurs at an early age, from pneumonia. Developmental defects in other organs are

common accompaniments of pulmonary agenesis; they include congenital heart disease, unilateral maldevelopment of the kidney, atresia of the anus, defects in the contralateral lung, and anomalies of the vertebral column. In the case reported by the author, there was defective development of the left diaphragm.

The patient was a 36-year-old female suffering from dyspnea and cyanosis on effort. Roentgen examination revealed a homogeneous density over the entire left hemithorax, which contained the stomach and left colon, with absence of the left diaphragm. Since bronchoscopy and bronchography were refused by the patient, tomography was resorted to; it revealed no evidence of a left main stem bronchus.

Five roentgenograms. W. C. GALLO, M.D.
Indiana University

Late Postoperative Pulmonary Atelectasis: A Syndrome of Late Spastic Atelectasis of the Left Lower Lobe, Associated with Acute Segmental Colonic Dilatation. Mariano R. Castex and Egidio S. Mazzei. Prensa méd. argent. **35:** 1577–1582, Aug. 13, 1948. (In Spanish)

Acute pulmonary collapse may involve all or part of a lobe or a lung. The involved zone loses its gaseous content and is reduced considerably in volume. The two fundamental mechanisms are exogenous compression and bronchial obstruction. This mechanism may act to produce an acute atelectasis (bronchitis, pneumonia, hemoptysis, injection of contrast substances in the bronchi) or as a chronic process (bronchiectasis, tumors, tuberculosis). Acute collapse is indicated subjectively by pain at the level of the affected hemithorax, with superficial respiration, and dyspnea of variable intensity; objectively it is manifested by retraction of the corresponding thoracic wall, with limitation of expansion and at times dislocation of the mediastinum toward the affected side, and by weakness or absence of sounds of vesicular breathing and resonance of the voice.

Radiologically one sees a homogeneous opacification of variable density, dislocation of the heart and mediastinum toward the affected side, pendulum movement of the heart and mediastinum in rhythm with respiration, depression of the thoracic wall, diminution of the intercostal spaces, elevation of the diaphragm on the affected side, diminution or repression of respiration.

This paper is limited to a discussion of the postoperative atelectasis which may supervene in a patient with normal respiratory apparatus. Postoperative atelectasis may occur immediately after any kind of surgical intervention, no matter what the type of anesthesia. Immediate causes include diminished pulmonary ventilation, inadequate bronchial drainage (modified by certain conditions due to the effect of the anesthetic on the patient), position of the patient during the operation, and type of surgical intervention. An abdominal incision often disturbs the abdominal muscles which take part in normal respiration and reduces the vital capacity and the effectiveness of cough. Spinal anesthesia may cause motor paralysis as high as the fourth thoracic segment, with paralysis of some of the intercostal muscles and reduction of pulmonary ventilation, with resulting bronchial stagnation.

The authors cite 30 cases; postoperative atelectasis appeared in 9 cases on the first day, in 13 cases on the second day, in 6 cases on the third day, and in 1 case each on the fourth and fifth day.

A further consideration involves bronchial constriction, which has been studied under the name of "spastic atelectasis," or "reflex atelectasis." The authors report 2 cases observed following cholecystectomy for gallstones, which differ somewhat from any previously reported, in the coexistence of atelectasis of the left lower lobe with acute functional dilatation of the colon in individuals with chronic constipation. Neither patient had suffered from any anxiety before the surgical operation, nor from a cold or acute or chronic bronchitis. Nor was there any evidence of infection in the mouth or pharynx. Operation was done under general gas anesthesia, with no unusual difficulty. The postoperative course was normal until the ninth day in one case and the fifteenth in the other, when there developed an acute functional dilatation of the colon accompanied by pulmonary atelectasis involving the left lower lobe. The clinical subjective, functional objective, and radiological pictures of pulmonary atelectasis were absolutely typical. In each case involution occurred within four or five days without any special treatment for the atelectasis, the treatment being directed only to the acute functional dilatation of the colon. Expectoration was not present at the onset, at the peak, or in the decline of the atelectatic process.

Differential diagnosis had to be made from pulmonary embolism or infarct; in neither of these cases was there any antecedent phlebothrombosis in the extremities, sanguineous expectoration, or pulmonary dulness prolonged over several days. On the contrary, the pulmonary process was afebrile.

The authors believe that the pathogenesis of these two cases was directly connected with the acute functional dilatation of the colon by stimulation of the sympathetics, for the following reasons: (1) functional disturbances of the colon usually affect that portion of the large bowel lying in the left upper quadrant of the abdomen; (2) the left lower lobe is most directly in contact with this section of the colon; (3) complete involution of the atelectasis followed treatment exclusively and effectively directed toward the abnormal colon.

Two roentgenograms. JAMES T. CASE, M.D.
Chicago, Ill.

Radiological Determination of the Level of the Diaphragm in Emphysema. M. Grossmann and H. Herxheimer. Brit. J. Radiol. **21:** 446–448, September 1948.

There has been heretofore no accurate method of indicating the degree of emphysema from roentgen examination, though this is often of considerable importance. A method is offered which will give a reliable indication of the diaphragmatic elevation and excursion, indicating thereby the degree of emphysema. Fifty-eight subjects were measured by this method, 8 normal, 5 obese, 7 with heart disease, and the remainder with clinical evidence of emphysema.

Three measurements are made on each patient; the maximum height of the dome of the diaphragm above the iliac crest at inspiration and at expiration and the length of the trunk (distance from the tuberosity of the ischium to the acromion process). The measurements of the diaphragm heights are best made at the fluoroscope, as the end points of the excursion can be observed. Sometimes the diaphragm is flat at inspiration. In this case the measurement is made to the point perpendicularly under the crest as observed at expiration.

From these measurements, two quotients are obtained: the ratio of the highest level of the diaphragm to the length of the trunk; the ratio of the lowest level of the diaphragm to the length of the trunk.

The mean quotients for the normal subjects were: high level 0.47; for the low level 0.35. For the patient with slight emphysema the mean quotients were high 0.44; low 0.31. In the patients with moderate and severe emphysema the high was 0.34 and the low 0.27. There was excellent correlation throughout the series. The obese patients and those with heart disease were within normal limits.

One table.

SYDNEY J. HAWLEY, M.D.
Seattle, Wash.

Cystic Disease of the Lung. Edward J. McGrath and Marcus J. Magnussen. Arch. Surg. **57:** 427–433, September 1948.

The authors report 15 cases of cystic disease of the lung, in all of which medical aid was sought because of problems directly related to the disease. Duration of symptoms was from five weeks to thirteen years, and the age range from three and a half to fifty years. Various initial diagnoses had been made, but in only one case did roentgen examination fail to produce confirmatory evidence of cystic disease. Cysts filled with air alone and those which had been evacuated by operative drainage appeared on the roentgenogram as spherical or ovoid areas delineated by a thin but well defined opaque wall, beyond which the pulmonary tissue was comparatively normally translucent. The cysts tended to retain their spherical or ovoid contour in both anteroposterior and lateral projections whether air-bearing, fluid-filled, or both. When the fluid-filled cyst was aspirated, it was found helpful diagnostically not to attempt complete evacuation and to replace the fluid withdrawn partially with air. Then, by positional roentgen studies, including those in lateral decubitus, it was possible to outline the cyst satisfactorily. Finally, where the cystic nature of the lesion was not obvious in routine roentgen studies, bronchography was helpful, showing elongation and displacement of the bronchi by the cyst.

Four tables.

LEWIS G. JACOBS, M.D.
Oakland, Calif.

Medical Surveys for Pulmonary Tuberculosis. Sidney A. Britten and Carl J. Heisser. U. S. Nav. M. Bull. **48:** 632–641, July-August 1948.

Continuing previous studies by Britten and his associates (see, for example, U. S. Nav. M. Bull. **46:** 936, 1946), the authors emphasize the usefulness of chest surveys in the military service, pointing out that if preservice films had been available for all personnel, and if the interpretation of those films had been perfect, the number of cases which were picked up later on would have been cut to half.

The present study covers 4,234 survey reports and includes an analysis of the findings of the boards of medical survey, with an attempt to evaluate the EPTE (existing prior to enlistment) status of the examinees.

It is pointed out, also, that further clinical and radiological study is necessary on those patients who show evidence of suspicious lesions by the survey method.

S. F. THOMAS, M.D.
Palo Alto, Calif.

Closure and Healing of Tuberculous Cavities. John Loesch. Am. Rev. Tuberc. **58:** 322–334, September 1948.

The author reports postmortem studies on four patients in whom roentgenographic observation had previously demonstrated the closure of tuberculous cavities (five cavities in all). In two instances the cavities were found to have been converted into closed inspissated foci containing variable amounts of calcium. These had been treated by pneumothorax. It is probable that cavities which close by being transformed into inspissated foci will remain as such for the duration of the patient's life. Though they may shrink in size over a period of years, their replacement by scar tissue probably takes place but rarely.

Eight roentgenograms; 1 photograph; 7 photomicrographs; 1 schematic drawing; 1 table.

L. W. PAUL, M.D.
University of Wisconsin

Accuracy of Roentgen Determination of Activity of Minimal Tuberculosis. Carl C. Birkelo and Paul O. Rague. Am. J. Roentgenol. **60:** 303–314, September 1948.

Seven hundred and sixty-five cases reported roentgenologically as minimal tuberculosis by the Detroit Department of Health were followed for two to five years to determine the accuracy of the original diagnosis. Criteria of activity during this period were (1) change in the roentgen appearance of the lesion; (2) positive sputum; (3) hospitalization; (4) therapeutic procedures; (5) death.

Excluding 66 cases originally reported as of "questionable activity" the activity was correctly determined from the original roentgenogram in 86.43 per cent of the cases. Of 572 cases originally reported as "minimal inactive tuberculosis," 489 remained inactive during the follow-up period, while of 127 cases reported as "minimal active," only 13 showed no activity. The percentage of accuracy for the first group was thus 85.5 and for the second 89.75.

An analysis of the 83 "mistakes" among the cases originally called inactive yielded little useful information. In four instances it was felt that the initial reading had been incautious. In two more the error was believed to result from attempting to determine activity from the 4 × 5-inch film. In 77 cases it was felt that the initial film would still be read as "inactive." It was noted that 24 per cent of these "errors" became evident some time after the second year of observation, indicating that inactivity for two years cannot be considered entirely "safe," and that follow-up at regular intervals must be continued for an indefinite period.

Sixteen roentgenograms; 4 tables.

ROBERT LARNER, M.D.
Baltimore (Md.) City Hospitals

Loeffler's Syndrome and Pulmonary Infiltrations Accompanied by Peripheral Eosinophilia. John C. Ham and Walter T. Zimdahl. Ann. Int. Med. **29:** 488–509, September 1948.

Preliminary to the presentation of their own cases, the authors review Loeffler's observations on the syndrome which bears his name, calling attention to the five characteristics which he emphasized: (1) infiltrations shown by roentgenogram, (2) fleeting and changing

character, (3) eosinophilia, (4) mild degree of illness, (5) short duration.

Three cases showing pulmonary infiltrations and blood eosinophilia are reported. The first case had a long stormy course which did not resemble the typical Loeffler syndrome. In the second case the course was less severe but quite protracted, with an onset suggesting angina pectoris. The third had a prolonged course with two episodes of pulmonary infiltration and eosinophilia; it was the only one of the three in which there was an allergic background.

The combination of pulmonary infiltration and eosinophilia may appear in any age group, from infancy to old age. In some cases the course has been very stormy, as illustrated by the authors' first case. The condition has occurred in association with definite asthmatic tendencies or other specific allergies, with *Endameba histolytica* infection and other parasites such as *Fasciola hepatica*, *Ascaris lumbricoides*, and trichinosis, with cutaneous helminthiasis and with brucellosis. It is thus an unusual response of the body tissues occasionally seen in many different conditions, with or without the manifestations generally considered to be allergic.

Periarteritis nodosa, Hodgkin's disease, and eosinophilic leukemia must be considered in the differential diagnosis, but usually can be ruled out by the clinical course. Pulmonary tuberculosis must also be considered, and with this in view the patient should be carefully followed.

Eight roentgenograms; 3 tables.

STEPHEN N. TAGER, M.D.
Danville, Ill.

Recurrence of Coccidioidal Cavities Following Lobectomy for a Bleeding Focus. David Krapin and Francis J. Lovelock. Am. Rev. Tuberc. **58:** 282–290, September 1948.

Persistent hemorrhage is considered one of the indications for lobectomy in coccidioidomycosis. The authors report a case in a 21-year-old white male with a cavity in the apex of the right lung. Because of recurrent bleeding, pneumothorax was instituted, but this failed to cause collapse of the cavity because of adhesions. Lobectomy was done and recovery from the operation was uneventful. Approximately a year later there was a recurrence of the disease, with cavitation in the remaining lung on the right. This was treated by thoracoplasty, which resulted in a satisfactory collapse. It is felt that this latter procedure is to be preferred over lobectomy when closure of coccidioidal cavities is required.

Two roentgenograms; 1 photomicrograph.

L. W. PAUL, M.D.
University of Wisconsin

Intracranial Metastases of Primary Pulmonary Carcinoma: A Diagnostic Difficulty. Michael B. Shimkin. California Med. **69:** 224–229, September 1948.

Carcinoma of the lung metastasizes more frequently to the brain than any other neoplasm, the incidence of cerebral metastases in reported autopsy series being 15 to 30 per cent. The spread is probably *via* the blood stream. Because the predominant symptoms may be referable to the central nervous system, these lesions may present a difficult diagnostic problem. Roentgen examination of the chest will show the presence of a pulmonary change in some 95 per cent of the cases, suggesting further studies upon which a diagnosis may be based.

Two cases are presented in which the symptoms and signs were primarily those of the intracranial lesion and the chest roentgenograms were interpreted as indicating tuberculosis. Even the operative diagnoses were erroneous, a diagnosis of papilloma of the choroid plexus being made in one case and of fibrous astrocytoma in the other. Only at necropsy was a correct diagnosis reached—primary lung carcinoma with intracranial metastases.

Two roentgenograms; 3 photomicrographs.

MAURICE D. SACHS, M.D.
Cleveland, Ohio

Surgical Removal of Unsuspected Mediastinal Lymphoblastomas: Report of Four Cases and a Review of the Literature. Byron H. Evans and Cameron Haight. Arch. Surg. **57:** 307–323, September 1948.

Single unilateral circumscribed mediastinal tumors of lymphoblastic origin are infrequently encountered. Roentgenologically they closely resemble dermoids and teratomas. A preliminary test dose of radiation prior to operation may provoke a significant response, but in some cases regression is not sufficient to indicate the diagnosis.

Four cases are reported. In all, the preoperative impression was dermoid cyst, though in one the possibility of lymphoblastoma was suggested. In none was a trial dose of radiation administered. In two the operative diagnosis was Hodgkin's disease and in two lymphosarcoma. One patient—with Hodgkin's disease—died more than seven years after operation. The others were alive at the time of the report, apparently without recurrence, after periods of five, four, and two and a half years.

Six previously reported cases of mediastinal Hodgkin's disease with apparent complete removal are reviewed.

Fifteen roentgenograms; 4 photomicrographs.

LEWIS G. JACOBS, M. D.
Oakland Calif.

Pulmonary Adenomatosis. A Report of Three Cases. George W. Drymalski, J. Robert Thompson, and Henry C. Sweany. Am. J. Path. **24:** 1083–1093, September 1948.

Three cases of pulmonary adenomatosis diagnosed at operation or autopsy are reported. One had undergone malignant change.

There are no distinguishing clinical features of pulmonary adenomatosis. Roentgenologically the condition simulates tuberculosis, carcinoma, or pneumonia.

One photograph, 5 photomicrographs.

Pulmonary Vascular Obstruction Due to Sarcoid. William Harbin, Jr., and Edward Bosworth. J. M. A. Georgia **37:** 337–340, September 1948.

Since it is not generally known that sarcoid can cause sufficient pulmonary vascular obstruction to lead to right heart failure, the authors report this case.

The patient was known to have sarcoid over a period of five years during which there was a gradual development of right heart failure, eventuating in death. At autopsy the lungs were found to contain enormous numbers of microscopic sarcoid nodules. Other con-

ditions commonly confused with sarcoid were ruled out.

Three illustrations, including 1 roentgenogram.

ZAC F. ENDRESS, M.D.
Pontiac, Mich.

Syphilitic "Tumor" of the Right Bronchus. Case Report. A. R. Judd. Ann. Otol., Rhin. & Laryng. **57:** 858–868, September 1948.

The authors present a case of syphilitic tumor (gumma) arising in the right stem bronchus. The patient was a 47-year-old man with a four-plus Wassermann reaction, complaining of difficulty in breathing and a cough productive of mucus and mucopurulent sputum, but no hemoptysis. The admission roentgenogram was suggestive of atypical pneumonia, showing right hilar density with nodular infiltration in the lower half of the right lung. A second film obtained three weeks later showed complete obliteration of the right lung field up to the second rib anteriorly and above this level a diffuse infiltration slightly more dense than that formerly seen in the lower part of the lung. The trachea, heart, and mediastinum were drawn to the right. The interpretation was "an atelectatic lesion due to bronchial obstruction." A bronchoscopic examination was then done, revealing a mass of tissue filling the right main bronchus and extending into the lower trachea. As much of the mass was removed as bleeding permitted, with a presumptive diagnosis of primary bronchial carcinoma. A week later most of the remaining tissue was removed and a microscopic diagnosis of syphilitic granuloma was made. A film obtained following removal of the obstructing mass and institution of antisyphilitic therapy showed absorption of the previous infiltration and renewed aeration over the right lung field, with little tracheal shift. Marked clinical improvement was also noted.

This case is believed to represent a unique occurrence, without counterpart in the literature. Distant syphilitic lesions were apparently absent except that the aorta appeared to show an increased arteriosclerotic change with saccular enlargement of the ascending portion.

Three roentgenograms; 1 drawing.

STEPHEN N. TAGER, M.D.
Danville, Ill.

Amebic Pleural Effusion. Case Report. Gordon McHardy and Donovan C. Browne. Gastroenterology **11:** 364–366, September 1948.

An interesting instance of amebic pleural effusion, assumed to be embolic from an intestinal focus without other demonstrable extra-intestinal amebiasis, is reported. The possibility of an amebic interstitial pneumonitis could not, however, be entirely ruled out.

Analysis of Malformations of the Heart Amenable to a Blalock-Taussig Operation. Helen B. Taussig. Am. Heart J. **36:** 321–333, September 1948.

The Blalock-Taussig operation is designed to increase the circulation to the lungs in persons who have an inadequate pulmonary blood flow. The objective is to place a minimal strain on the heart and to give an adequate, not excessive, pulmonary blood flow.

Six criteria have been established to determine the advisability of operating for any unusual cardiac malformation: (1) the primary difficulty must be lack of adequate pulmonary blood flow; (2) a suitable pulmonary artery must be present to which to anastomose the systemic artery; (3) a systemic artery must be available for anastomosis; (4) there must be a sufficient difference in pressure between the systemic and pulmonic circulations so that blood will flow from the aorta to the pulmonary artery; (5) the structure of the lungs must be such that the patient can survive the collapse of one lung and the occlusion of one pulmonary artery; and (6) the structure of the heart must be such that it can adjust to the altered circulation.

Experience has shown that patients with a tetralogy of Fallot can adjust to the changes in the circulation following operation. Other types of malformation which have been improved are those with a cardiac contour similar to that of a tetralogy of Fallot with left axis deviation; partial rotation of the heart on its axis; possibly "pure" pulmonary stenosis and an auricular septal defect; and a few unusual arrhythmias.

Progressive cardiac enlargement following surgery has been rare. About 30 per cent of the patients have shown no demonstrable cardiac enlargement, while another 30 per cent have shown some enlargement during the first three weeks after operation but none thereafter. Thirty per cent showed some cardiac enlargement during the first six months following surgery but no further increase.

Present evidence indicates that in most instances an adequate pulmonary blood flow can be attained by the use of the subclavian artery for the anastomosis, and that the degree of enlargement is less than when the innominate artery is used.

The author discusses the density of the hilar shadows in relation to collateral circulation and the value of angiography for demonstration of the pulmonary artery

Four tables.

HENRY K. TAYLOR, M.D.
New York, N. Y.

Congenital Tricuspid Atresia Associated with Interauricular and Interventricular Septal Defects. J. B. Miale, A. L. Millard, T. J. Beno, and G. S. Custer. Am. Heart J. **36:** 438–442, September 1948.

The authors report in detail a case of congenital tricuspid atresia, with associated interauricular and interventricular septal defects, including the necropsy findings.

Tricuspid atresia should not be confused with other congenital anomalies. In the cyanotic group, it is the only lesion which gives a left axis deviation and is thereby easily distinguished from cases of tetralogy of Fallot. Also, the roentgenogram is characteri showing absence of the conus shadow.

Tricuspid atresia is relatively rare. This is said to be the forty-first case to be reported.

One roentgenogram; 3 photographs.

HENRY K. TAYLOR, M.D.
New York, N. Y.

Movement of the Mitral Ring in Cases of Pathologic Changes in the Left Ventricle Especially Within the Posterior Papillary Muscles. Henning Odqvist. Acta radiol. **30:** 182–190, Sept. 30, 1948.

The author has studied the movement curves of calcified cardiac valves and annulus fibrosus by means of cineroentgenography and concludes that the condition of the heart muscle, as well as the localization of the calcification, determines the movement curve.

If the annulus fibrosus of the mitral valve is calcified and the heart is otherwise unimpaired, the mitral ring goes down and up in a rectilinear course. If there are pathologic changes in the posterior group of papillary muscles, including the posterior ventricular wall, a lack of co-ordination occurs in the muscular contractions, and the course of movement is triangular (at the end of systole, the mitral ring moves anterolaterally).

Seven roentgenograms; 2 drawings.

M. M. MANALAN, M.D.
Indiana University

THE DIGESTIVE SYSTEM

Radiological Aspect of the Normal Esophagus in Nursing Infants. M. Malenchini, J. Roca, and T. Banzas. Prensa méd. argent. **35:** 1698–1699, Aug. 27, 1948. (In Spanish)

To study the normal esophagus in the infant, the authors administered the opaque medium (diluted as much as possible without endangering its opacity) with an ordinary nursing bottle and examined the child in the vertical position.

The filled esophagus appeared as an opaque band occupying the retrocardiac space. It differed from the adult esophagus only in its narrower caliber. As age advances this difference disappears, and in the last months of the first year of life the shadows are practically similar in the infant and in the adult.

The tendency of the nursing child to swallow air is well known and the bubbles of air passing down the esophagus should not cause errors of interpretation. During swallowing there is in many cases a widening of the extremity of the esophagus just above the hiatus. This area corresponds to the phrenic ampulla of the adult and the finding should not be considered pathological.

Five roentgenograms. JAMES T. CASE, M.D.
Chicago, Ill.

Diverticula of the Stomach. Carlos Bonorino Udaondo, Manuel A. Casal, and Victorino D'Alotto. Prensa méd. argent. **35:** 1759–1765, Sept. 10, 1948. (In Spanish)

Up to 1937, 106 cases of gastric diverticula had been published. The authors have found 9 cases in recent years at the National Institute of Gastroenterology in Buenos Aires. Seventy-five per cent of these diverticula were in the infracardiac region or in the upper pole of the stomach. They are rounded and variable in size. Some are wide and have a short pedicle; others have a narrow communication which is scarcely discernible. The regional rugal folds converge toward the site of the diverticulum. The fundus is generally found free in the peritoneal cavity. Diverticula in the pyloric zone are larger. Those localized on the lesser and greater curvatures are rare. Endoscopy is of little diagnostic value in most cases. The circular diverticular orifice of the diverticulum is usually covered by marginal rugous folds.

Eleven roentgenograms. JAMES T. CASE, M.D.
Chicago, Ill.

Direct Demonstration of Perforated Ulcers. J. Frimann-Dahl. Acta radiol. **30:** 177–181, Sept. 30, 1948.

In 268 cases of perforated ulcer, the plain roentgenograms showed a niche in 8 per cent. Some of the diagnoses were made in retrospect, and some preoperatively. The gas in the stomach acts as a contrast medium, filling the crater, which may be fairly well demonstrated on the film. The ulcers most readily seen were those on the lesser curvature at or above the incisura or in the duodenal bulb. The best position for their demonstration was in left lateral recumbency. In this position a pneumoperitoneum also appears distinctly, since the gas moves toward the pyloric region with the greatest chance of penetration into the abdominal cavity. In other cases the gas may fill the niche and be fairly well demonstrated in the ordinary upright or even the supine position.

Five roentgenograms. R. DATZMAN, M.D.
Indiana University

An Unusual Complication of Miller-Abbott Intubation. Report of a Case. Joseph E. Caruolo. New England J. Med. **239:** 396–397, Sept. 9, 1948.

A case is reported in which an attempt to withdraw a Miller-Abbott tube introduced for therapeutic purposes led to serious respiratory difficulties and moderate cyanosis. Inspection of the pharynx showed within it a knotted mass of tubing lying against the posterior wall. The case emphasizes the importance of roentgen and fluoroscopic control of intubation, which would certainly have prevented the complication.

One photograph.

Tuberculosis of the Stomach and Duodenum. Herman W. Ostrum and William Serber. Am. J. Roentgenol. **60:** 315–322, September 1948.

Tuberculosis of the stomach and duodenum is uncommon. The pathogenesis is discussed and the following probable routes of infection are mentioned: (1) direct infection through the mucosa; (2) the blood stream; (3) the lymphatics; (4) spread from contiguous structures, especially the lymph nodes. Although there have been many theories advanced to explain the rarity of the condition, none has been proved.

The gastric lesion is most often ulceration, single or multiple, usually occurring along the lesser curvature in the prepyloric region. The second most common lesion in the stomach is the proliferative or hypertrophic type, with invasion of the gastric wall, often with tumor formation. This lesion is most often confused with carcinoma. All varieties of lesions have lymph node involvement, which may dominate the picture.

The duodenal lesion may be the result of a gastric lesion which has invaded through the pylorus into the duodenum. The second or third part of the duodenum may be involved. This occurs more frequently in tuberculosis than in peptic ulcer.

Tuberculosis of the stomach and duodenum may occur with or without tuberculosis elsewhere in the body. Approximately 10 per cent of the cases are associated with gastric carcinoma.

The roentgenographic findings are not pathognomonic, though some authors have reported findings which they believe to be suggestive.

The authors present three cases in which the chief pathological characteristic was the presence of ulcerations and fistulous tracts. Although fistulous tracts were not diagnosed roentgenologically in these cases, visualization of such fistulae should make the examiner suspicious of tuberculosis. Some of the findings other than fistulous tracts which may lead to a suspicion of

tuberculosis are extension of a lesion from the stomach to the duodenum and evidence of extrinsic pressure upon the digestive tract due to enlargement of the gastric lymph nodes.

Ten roentgenograms. EUGENE R. KUTZ, M.D.
Baltimore (Md.) City Hospitals

Failure of Rotation of Mid-Gut Loop. Gregory L. Robillard, William J. Fusaro, and Celso R. Garcia. Am. J. Surg. **76:** 332–337, September 1948.

The authors report failure of rotation of the mid-gut loop, as seen in a 14-year-old white boy. This case is unique in that the patient had had no digestive symptoms up until one week before admission. When first seen, he was complaining of severe epigastric and lower abdominal pain and nausea. Soon afterward he began to vomit undigested food particles forcefully. Examination revealed lower abdominal tenderness, most marked over the right lower quadrant. The temperature was 100° F., pulse 84, respirations 20, white blood cell count 15,000 with 82 per cent polymorphonuclears. A diagnosis of acute appendicitis was made and surgery was performed. At operation, the small intestine was found in the upper abdomen, encapsulated in a mass the size of a football. This was reduced and recovery was uneventful until the tenth postoperative day, when the patient experienced a recurrence of severe epigastric pain and again began to vomit undigested food. An emergency laparotomy revealed the small bowel involved in an internal herniation which was causing intestinal obstruction. Exploration at this time showed the cecum to be anomalously placed in the epigastric region overlying the bodies of the dorsal vertebrae. The ascending and transverse colon were situated along the left border of the dorsal vertebrae from above downward toward the pelvis, then extending upward to join the descending colon at the splenic flexure. The descending and sigmoid colon were normally placed. Numerous adhesions were found in the region of the ligament of Treitz. The herniation was reduced; an appendectomy, with lysis of numerous adhesions, was performed. The patient made an uneventful recovery except for a brief episode of influenzal meningitis, which rapidly subsided. A gastro-intestinal series made before discharge from the hospital revealed the anomalous position of the cecum, ascending and transverse colon. The films are reproduced to good advantage.

The authors give a brief review of the literature and point out that failure of rotation of the gut in the second stage often produces volvulus shortly after birth, when the motor activity of the intestine sets in. This diagnosis should be suspected when vomiting occurs in the first few days of life in contradistinction to pyloric stenosis, in which the onset is usually after a week. In addition, there is no bile in the vomitus in pyloric stenosis. X-ray examination will help determine whether the difficulty is on the basis of duodenal atresia. In a smaller group of patients symptoms may not appear till later in life.

Four roentgenograms. PAUL W. HOFFERT, M.D.
University of Pennsylvania

Small Intestinal Deficiency Pattern: Current Status. W. H. Glass. Am. J. Digest. Dis. **15:** 294–298, September 1948.

This paper is essentially a review of published work on the changes demonstrable radiologically in mucosa of the small intestine, described by Golden as due to a deficiency of vitamin B complex and shown by others to occur in a variety of conditions, including non-tropical sprue, steatorrhea, celiac disease, etc., and even in association with severe emotional upsets.

The author reports a series of 10 cases on the basis of which he concludes that "the entity of the small intestinal deficiency pattern is early reversible idiopathic steatorrhea." He is in agreement with Golden that the term "small intestinal deficiency pattern" should be discarded in favor of the term "disordered motor function," with its etiologic basis.

It is emphasized, also, that a normal small intestinal pattern covers a wide range of latitude which may coincide or vary greatly from our idea of normal, with the patient showing no abnormal symptoms to account for the changes.

Four roentgenograms, 1 table.

JOSEPH T. DANZER, M.D.
Oil City, Penna.

Primary Malignant Tumours in the Small Intestine, with Special Reference to Their Roentgen Diagnosis: A Survey and a Report of One Case. Rolf Köhler. Acta radiol. **30:** 217–224, Sept. 30, 1948.

Primary malignant tumors of the small intestine are rare, occurring in only 0.1 per cent of a routine autopsy series. Indefinite pains, sensations of pressure below the chest, a sense of overfullness, and vomiting are among the symptoms, generally gradual in onset though occasionally they appear rapidly. Pathologically, the tumors may manifest themselves as constricting, fungating, or diffusely infiltrating lesions. Administration of a barium-water mixture by mouth or through a Miller-Abbott tube may reveal the following signs of malignancy: obliteration of the folds of the mucous membrane in the entire length of the lesion, which shows rough, jagged contours; an eccentric lumen through an inflammatory lesion that is concentric with the lumen of the uninvolved segments of the bowel; a short, sharply delimited, marginal or circular deformity; stiffness of the affected segment, which moves in its entirety when shifted.

The author presents a case report of a 71-year-old woman with symptoms of two to three weeks duration. A constrictive lesion was demonstrated roentgenographically 15 cm. distal to the duodenal-jejunal flexure. At surgery an adenocarcinoma was removed. Eighteen months later the patient was in good condition and roentgenographic examination normal.

A roentgenogram of the lesion and a photograph of the surgically excised specimen are reproduced.

The author enumerates the other cases of roentgenologically diagnosed malignant tumors of the small intestine reported in the Scandinavian literature.

P. B. LOCKHART, M.D.
Indiana University

Calcified Mucocele of the Appendix. J. Peyton Barnes. Am. J. Surg. **76:** 323–327, September 1948.

The article is a case report of an asymptomatic calcified cyst in the region of the cecum which was first noted on physical and radiological examination in reference to gallbladder disease. The calcified mass was removed at the time of the cholecystectomy and was found to be in contact with the cecum at the normal site of the appendix. No appendix was found. The pathological report described the cyst as a calcified mucocele of the appendix.

Six roentgenograms; 1 photograph.

JOHN F. WEIGEN, M.D.
University of Pennsylvania

Spontaneous Fistula Between the Biliary and Digestive Tracts. Chronic Pyloroduodenal Occlusion by a Biliary Calculus. Alejandro J. Pavlovsky and J. Alfredo Ferreira. Prensa méd. argent. **35:** 1628–1635, Aug. 20, 1948. (In Spanish)

Pyloroduodenal occlusion due to a gallstone is a rare complication of duodenobiliary fistula. The diagnosis is rarely made, the patient usually reaching the surgeon after a long illness, which accounts for the relatively high mortality in these cases. It is possible from the symptoms to suspect the presence of the lesion, but in most cases the diagnosis can be made only after x-ray study. There are typical signs which should point to duodenobiliary fistula, such as the presence of gallstones in the vomitus or the passage of a large gallstone per rectum without any previous objective phenomena, or intestinal obstruction in known cases of cholelithiasis. Biliary lithiasis can produce true pyloroduodenal stenosis by constrictive perivisceritis or by lodgment of a calculus. Complete occlusion of the intestinal tract is compatible with only a few days of life. High obstruction of the duodenum may be tolerated for weeks or even months. It is important to remember that duodenal occlusion may present itself with typical cholelithic attacks, it may be preceded by vague disturbances, or it may occur without any previous symptoms.

Two important signs may be observed either independently or in association. There may be remission of painful symptoms on the appearance of the pyloroduodenal syndrome. The migration of the stone into the duodenum, at the same time that it sets up an occlusion, diminishes the tension in the biliary tract, and this causes disappearance or attenuation of the painful crisis. The second sign consists of hemorrhage, which usually appears during a frank painful crisis or immediately after it passes, due to erosion of intestinal vessels produced by movement of the stone.

The radiologic diagnosis of fistula between the biliary and digestive tracts is based on partial or total air visualization of the biliary passages or on the gas shadow surrounding a stone as seen in simple films, or on the spontaneous or provoked penetration of barium into the biliary tract. One should recall that cholecystitis due to an aerobic organism may be interpreted radiologically as a gas shadow. It should be recognized by the gaseous image of the gallbladder and by intraparietal or perivesicular bubbles. Similarly the penetration of barium into the biliary tract may be observed in case of a destructive lesion of the papilla of Vater. On the contrary, the anatomical characteristics of fistula may oppose or may hinder the penetration of air or barium even when one tries forced passage of air by deliberate manipulations. This succeeds in a fair percentage of cases, especially in occlusion due to calculi, when the stone not only obstructs the intestines but also frequently occludes the fistulous passage.

Eight roentgenograms.

JAMES T. CASE, M.D.
Chicago, Ill.

Routine Use of Operative Cholangiography. Philip F. Partington and Maurice D. Sachs. Surg., Gynec. & Obst. **87:** 299–307, September 1948.

The authors report their results with the routine use of operative cholangiography on all patients requiring gallbladder or common duct surgery during the year 1946–47. Thirty cases constitute the series.

An attempt was made to eliminate the causes for poor films and delay. A plywood tunnel was placed beneath the abdomen, the biliary duct system is carefully inspected, and the cystic duct at its junction with the common duct is dissected. A tie is placed on the cystic duct close to the gallbladder to prevent injection of radio-opaque material into the latter. A quarter-inch 22-gauge needle is then inserted into the common bile duct. This needle is connected to a 30-c.c. syringe by means of a foot or more of amber rubber tubing. It is preferable to have the syringe and needle filled with saline to avoid the introduction of air bubbles and to check the position of the needle. After it is certain that the needle communicates with the lumen of the common duct, a syringe containing 70 per cent diodrast is substituted for the saline syringe. Twenty cubic centimeters of this opaque material are injected slowly to prevent sudden dilatation of the biliary tree. Of importance is preliminary testing of the patient for sensitivity to the diodrast by instillation of a drop of the medium into the conjunctival sac.

After the roentgenogram is made and during its processing, the surgeon can excise the gallbladder unless unavoidable indications for exploration of the common duct require this to be done first. Wet films are available for inspection and interpretation before the completion of either procedure. If common duct exploration is necessary, a repeat cholangiogram is taken through the T-tube before the abdomen is closed. The second set of wet films is usually ready for interpretation before the abdominal closure is completed. Any further exploration may be accomplished at the comparatively simple cost of removing a few sutures.

Although the objective of operative cholangiography as first used was the determination of the presence of common duct calculi, it has proved a diagnostic aid in other lesions involving the biliary duct system, such as tumors of the head of the pancreas, common duct, or liver; inflammation or spasm of the sphincter of Oddi; indirectly, pancreatitis or hepatitis. In order to obtain maximum proficiency with operative cholangiography, the procedure should be made routine in gallbladder surgery.

Fifteen roentgenograms, with accompanying drawings; 1 photograph.

MARLYN W. MILLER, M.D.
University of Pennsylvania

THE SPLEEN

Diagnosis of Splenomegaly. Alvaro Barcellos Ferreira. Prensa méd. argent. **35:** 865–875, May 7, 1948. (In Spanish)

It is not always possible by the usual methods of physical examination to determine with certainty the real size of the hypertrophied spleen. In such cases it is wise to resort to radiographic study. This includes fluoroscopy of the diaphragm, with analysis of its position and mobility; then simple radiography, antero-

posteriorly and left laterally; determination of the position and morphologic characteristics of the spleen and left kidney. One then proceeds to contrast examination of the digestive tube to determine the relation of the enlarged spleen to the stomach, the duodenojejunal angle, and the left flexure of the colon.

The diaphragm may be more or less elevated. The stomach is displaced more or less to the right anteriorly, depending on the size of the spleen, and is sometimes partially compressed. The duodenojejunal angle is not displaced forward anteriorly, although it may be displaced toward the midline. The colon is pushed downward and toward the left. JAMES T. CASE, M.D.
Chicago, Ill.

Solitary Calcified Cyst of the Spleen. Joseph A. Witter and Viola G. Brekke. Am. J. Surg. **76:** 315–318, September 1948.

Cysts of the spleen are classified as true or false depending on the presence of a specific lining membrane. False cysts, or pseudocysts, which have only a hyaline fibrous wall or a condensed rim of splenic tissue may be hemorrhagic or serous, inflammatory or degenerative. A case of the serous type is reported here.

The patient was a 48-year-old white woman who complained of fullness, a pressing sensation, and dull aching pain in the left upper quadrant of the abdomen, aggravated by eating. She had twice injured the left anterior chest wall, seventeen and twenty years previously, fracturing several ribs on the earlier occasion. Physical examination was essentially negative and laboratory studies revealed nothing but an increased prothrombin time. X-ray films of the abdomen showed a spherical, partially calcified tumor in the left upper quadrant 4 inches in diameter, just below the diaphragm. At operation a large cyst with a calcified wall was found in the upper pole of the spleen. The spleen was removed and the patient had an essentially uneventful recovery.

On section the cyst was found to be encapsulated and filled with thin reddish-brown fluid, glistening with cholesterol crystals. It measured 8 × 9 cm. and its lining was fairly smooth, pale yellow, and calcified except for areas of trabeculation between which the rim of splenic tissue was seen. Microscopically the wall was composed of dense hyalinized connective tissue and there was no epithelial lining.

Roentgenograms are of considerable value in the diagnosis of cysts of the spleen, especially if the wall is calcified. Indirect evidence evinced by downward displacement of the splenic flexure is also diagnostic. Ordinary splenomegaly does not displace the left colon.

Two roentgenograms; 2 photographs; 1 photomicrograph. ALLAN K. BRINEY, M.D.
University of Pennsylvania

THE MUSCULOSKELETAL SYSTEM

Chronic Inflammatory Lesions of Bone Resembling Neoplasms. Report of Three Cases. Lewis W. Breck, W. Compere Basom, M. S. Hart, and James R. Herz. Texas State J. Med. **44:** 386–389, September 1948.

Three interesting cases are presented, all of bizarre chronic inflammatory lesions of the bone, demonstrating neoplastic characteristics on the roentgenogram. The clinical histories, also, were confusing. In the first case, in a 13-year-old boy, the picture resembled Ewing's endothelial myeloma. In the second case, a chronic non-suppurative lesion in a 58-year-old man was first thought to be an osteogenic sarcoma. In the third patient, a 3-year-old boy, the condition was originally believed to be an osteolytic-osteogenic sarcoma.

The authors believe that it might be well for the Bone Tumor Registry to register these bizarre inflammatory lesions of the bone, as well as bone tumors, in the interests of better differential diagnosis. They also stress the importance of a careful biopsy before the institution of radical treatment in cases of suspected neoplasms of the bone.

Three roentgenograms. S. F. THOMAS, M.D.
Palo Alto, Calif.

Endothelioma of Bone (Ewing's Sarcoma). Bradley L. Coley, Norman L. Higinbotham, and Lemmel Bowden. Ann. Surg. **128:** 533–558, September 1948.

In the period from 1918 through 1947, only 91 histologically proved cases of endothelioma of bone, or Ewing's tumor, were seen in Memorial Hospital (New York). Even so, osteogenic sarcoma is the only primary bone tumor occurring more frequently. Males are more often affected than females. The average age of the patients in the series reported was fifteen years.

The signs, symptoms, and histologic appearance of this neoplasm suggest the possibility of virus infection as its cause, but the actual etiology remains obscure. Ewing's tumor is much more common below the waist, appearing most often in the femur. Pain, swelling, disability, and fever are the outstanding clinical manifestations.

The roentgenographic picture is typical, showing an osteolytic lesion of bone, irregular in outline, of central or subcortical origin, rapidly involving the entire circumference of the bone. The overlying periosteum may have an "onion-peel" appearance. Soft-part involvement may be seen. Unfortunately, 51 of the 91 cases here reported resembled other conditions, such as osteogenic sarcoma, reticulum-cell sarcoma, inflammatory disease, etc. The primary site is usually in the diaphysis of a long bone. Pathologic fracture was present in 21 per cent of this series.

The blood levels of calcium, phosphorus, and phosphatase are usually normal. Leukocytosis may occur but is not very marked. The histologic picture shows crowding of cells in some areas and relatively few cells in others. The cell has a round or oval nucleus with one or more nucleoli and often a poorly defined cell membrane. Leukocytic infiltration is common.

For the purpose of diagnosis, every case should have microscopic confirmation before the institutution of roentgen therapy, since the histologic picture may be entirely misleading after even mild exposures to radiation.

Neurologic disturbances associated with cerebral or spinal metastases occurred with surprising frequency in the authors' series. Early widespread metastasis is the rule, most often to the lungs, next to the other bones of the skeleton, and then to the lymph nodes and other viscera. The metastases possess the same characteristics as the primary tumor. The occurrence of fever in a patient who has had Ewing's tumor usually indicates metastasis or a local recurrence.

X-ray therapy, radium, surgery, Coley's serum, and in 3 patients radioactive phosphorus were used in the series reported. The end-results were poor. Only 3 of 73 patients seen between 1918 and 1942 survived five years or more. The average survival was 18.7 months.

Fifteen illustrations, including 11 roentgenograms; 8 tables.

PAUL W. ROMAN, M.D.
Baltimore, Md.

A Case of Yaws in New York City. Charles F. Post and Charles Sheard, Jr. New York State J. Med. **48:** 1920–1925, Sept. 1, 1948.

A case of yaws in a Negro boy in New York City, who had spent some years in Martinique, is reported. The emphasis is chiefly on the dermatologic features but the following roentgen findings were of interest. X-ray examination of the chest, long bones, hands, and feet revealed bilateral, small, rounded defects in the cortex of both humeri just distal to the proximal ephiphyseal line; questionable cortical destruction of the medial margin of the left tibia just below the knee joint, and an area suggesting destruction of bone in the midportion of the epiphysis of the proximal phalanx of the right great toe.

There was a favorable response to penicillin and subsequent roentgen examinations of the bone lesions revealed a tendency to slow healing.

Nine illustrations; 4 tables.

A Defect in the Second Lumbar Vertebra at the Junction of the Neural Arch with the Vertebral Body. Olle Olsson. Acta radiol. **30:** 243–248, Sept. 30, 1948. (In German)

The author reports the case of a 37-year-old woman in whom the second lumbar vertebra showed a failure of fusion at the junction of the neural arches with the vertebral body. On the lateral roentgenogram of the lumbar vertebrae, bilateral, smooth, slightly curved dehiscences, 1–2 mm. wide, were seen just posterior to the lateral portion of the vertebral body. There was normal calcification of the neural arches, with no evidence of any destructive process or of any new bone formation. The ossification center in the vertebral body normally fuses with the center in each arch between the third and sixth year.

The author emphasizes that the posterior lateral portion of the vertebral body is partly formed by the base of the neural arch. Therefore, the site of fusion, *i.e.*, the neurocentral suture, can be incorporated into the posterior lateral part of the vertebral body. In the lumbar vertebrae this suture line runs obliquely, whereas in the body of S1 it runs in the sagittal plane. These observations were based on radiographs made on anatomical vertebral columns of children up to six years of age.

This case, therefore, is considered as a failure of ossification at the site of the normal fusion of the neural arches with the vertebral body. An isolated finding such as this defect in a completely ossified spine has never been previously reported.

Three roentgenograms; 1 drawing.

SAMUEL MORCHAN, M.D.
Indiana University

On Axial Projection of the Shoulder-Joint. Folke Knutsson. Acta radiol. **30:** 214–216, Sept. 30, 1948.

Knutson recommends the axial projection of the shoulder joint for diagnosis of anterior or posterior subluxations of the humeral head. The films are made with the patient recumbent, with the arm abducted from the trunk at 90 degrees, and central rays directed through the axilla onto the film held above the shoulder. Absence of the double outline of the anterior glenoid margin indicates recurrent dislocation of the humeral head.

Nine roentgenograms, one photograph, one diagram.

J. A. CAMPBELL, M.D.
Indiana University

Evolution and Treatment of Tuberculosis of the Hip. Ignacio Ponseti. Surg., Gynec. & Obst. **87:** 257–276, September 1948.

This is a report of 31 cases of tuberculosis of the hip proved by guinea-pig inoculation of abscess material or tissue from the involved hip. All patients were followed for a minimum of four years. The cases are divided into three groups: (1) primary para-articular bone lesions, 7 children; (2) tuberculosis of the hip joint in childhood (under fifteen years of age), 15 patients; (3) tuberculosis of the hip joint in adults, 9 patients.

The author agrees with others that not the age but the date of primary infection regulates the type of reaction seen in osteoarticular tuberculosis. The most destructive lesions are seen, as a rule, in children, as contact with the tubercle bacillus usually occurs early in life. In addition, the capacity for repair changes with age. On these factors he justifies his classification.

(1) In the para-articular group two types of tuberculous osteitis are described. The first, designated granulous osteitis because of an abundance of tuberculous granulations, appears roentgenographically as an area of uniform bone destruction with only moderate sclerosis, or none at all, surrounding it. The second, called caseous osteitis because of the abundance of primary caseous necrosis in the marrow, shows an area of bone destruction containing one or more dense sequestra and surrounded by dense sclerosis.

In this group of cases both osteocopic and arthritic pain were present on admission and in every instance the hip joint was affected sooner or later, regardless of the course of the bone lesion. The type of reaction seen in the joint bore no relation to the type of preceding bone lesion. The cause for the predominance of one type is not stated, but it is suggested that it depends on varying allergy of the patient. The conclusions drawn are: (*a*) that extra-articular foci of osteitis are more common in children and are rare in adults; (*b*) the femoral head is almost never involved by primary foci of tuberculous osteitis; (*c*) the hip joint invariably becomes involved.

(2) In tuberculosis of the hip in children, the joint capsule sometimes appeared widened in roentgenograms taken in the early months, due to the accumulation of caseous debris within the joint capsule. When the joint capsule burst and the debris emptied, forming a soft-tissue abscess, the joint space became narrowed.

From a year and a half to three years after the onset of symptoms re-ossification of the atrophic bone occurred. This is the "healing reaction." The "re-ossification" is irregular, and a few dense bone trabeculae are seen crossing the atrophic area. The joint space remains narrow unless the patient is allowed to bear weight, in which case it will widen.

During the acute stage, soft-tissue abscesses are not seen on the roentgenogram, but when they become chronic they are usually visible.

The author recommends fusion of the hip after the "healing reaction" is well established, as only in "rare" exceptions is a stable, painless hip obtained by immobilization. The procedure is best done during the third year after onset.

(3) The adults with tuberculosis of the hip joint are divided into three groups. The first includes those patients with "cured" or minimal pulmonary tubercu-

losis; minimal signs and symptoms referable to the hip; slow, progressive thinning of the joint space; moderate destruction of subchondral bone of the femoral head and acetabulum, and arrest of the process in two to three years. These cases may be treated by extra-articular hip fusion.

The second group is composed of patients with active pulmonary tuberculosis in whom the hip lesion develops rapidly, with prompt narrowing of the joint space, extreme bone atrophy, and melting away of the joint or bone sclerosis. The hip lesion is considered a part of a generalized process in these cases and surgery is definitely contraindicated. The author's four cases of this group all terminated fatally.

The third group of the adult type was made up of patients with no, or minimal, pulmonary tuberculosis, but very destructive osteo-articular tuberculosis and large abscess collections. These cases are best treated conservatively until the destructive process is arrested.

Forty-seven roentgenograms; 5 photomicrographs.

PAUL R. NOBLE, M.D.
University of Pennsylvania

Insertion of the Smith-Petersen Pin. Martin L. Quinn. Am. J. Surg. **76:** 289–297, September 1948.

A method is described for the accelerated introduction of the Smith-Petersen pin in femoral neck fractures, with full details as to the preoperative, operative and postoperative management.

Anteroposterior and lateral radiographs are made to determine the position of the fracture, the position of the Kirshner guide wires at operation, and the position of the pin at or after operation.

Of fundamental interest to the radiologist are the following points, which are briefly discussed: (1) the influence of type and location of fractures on treatment and prognosis; (2) method and depth of insertion of the pin and pitfalls associated with improper pinning; (3) description of the blood supply of the upper end of the femur and the factors that influence healing; (4) positioning of the hip for satisfactory radiography; (5) radiographic criteria for satisfactory pinning and the complications incident to unsatisfactory pinning.

Ten roentgenograms; 1 drawing.

DANIEL TALLEY, III, M.D.
University of Pennsylvania

A Case of Osteochondritis Dissecans of the Ankle. Thor Narvestad. Acta radiol. **30:** 209–213, Sept. 30, 1948.

A 22-year-old man sustained direct trauma to his left foot. Radiographs taken immediately thereafter showed separation of a small fragment from the lateral edge of the superior articulating surface of the talus. This was considered to represent a fracture. Ten months later tenderness, pain, and limitation of motion persisted. Radiographs were then taken at the author's hospital, showing in the same part of the talus "bean-sized rarefaction and in this a sclerotic shell-shaped fragment about 1 cm. long. There was some slight sclerosis about the rarefied area."

Since the fragment of bone was present immediately after injury, the author concludes that an "already existing osteochondritis dissecans with superficial necrosis of the bone became active through the separating off of the fragment." No operation was performed.

Three roentgenograms.

R. LEVIN, M.D.
Indiana University

GYNECOLOGY AND OBSTETRICS

Estimation of Pelvic Capacity. William F. Mengert. J. A. M. A. **138:** 169–174, Sept. 18, 1948.

There are five components of cephalopelvic disproportion: (1) size and shape of the bony pelvis, (2) size of the fetal head, (3) force exerted by the uterus, (4) moldability of the head, and (5) presentation and position. Of these, only the first is susceptible to accurate measurement. Attempts to measure the size of the unborn fetal head have not met with much success. There are three imponderables: force of labor, moldability of the fetal head, and method of presentation, of which force of labor is most important.

The author's series includes 935 patients, of whom 592 had been delivered. The pelvis of each patient was measured manually and radiographically with Snow's technic.

Measurement of the anteroposterior and transverse diameters of a sufficient number of cross sections of the bony pelvic canal will provide knowledge of its capacity. Three cross sections—inlet, midplane, and outlet—are considered sufficient. Since the outlet consists of two triangles with a common base, and since the fetal head emerges through the posterior triangle, the posterior sagittal diameter of the outlet is used rather than the anteroposterior.

After employing many methods, the author arrived at one by which it is possible to determine the relation of one pelvis to another or of one plane to another. The simple expedient of multiplying the transverse and the anteroposterior diameters and expressing the capacity of the various planes in terms of the product was employed. This furnishes as satisfactory a measure of relative capacity as the more laboriously obtained measurement of the actual area. A normal can be established for each plane and its capacity expressed as a percentage of normal. The average value for the product of the anteroposterior and transverse diameters for the inlet was found to be 145 and for the midplane 124.9.

By a process of trial and error, it became obvious that 85 per cent of normal capacity of either inlet or midplane represented the borderline between adequacy and contraction. In general, inlet and midplane tended to vary together. It is doubtful whether outlet contraction exists unassociated with midplane contraction.

Criteria of suspicion indicating the necessity for employment of radiographic pelvic measurement previously published (Eller and Mengert: Am. J. Obst. & Gynec. **53:** 252–258, 1947. Abst. in Radiology **49:** 754, 1947) are repeated here.

Twelve diagrammatic drawings and charts.

S. B. FEINBERG, M.D.
University of Michigan

Hysterosalpingography. Raymond Simard and Georges Fortier. Canad. M.A.J. **59:** 220–224, September 1948.

Hysterosalpingography is a simple, innocuous procedure, which can be carried out on the ambulatory patient, and may at times be an indispensable aid in the diagnosis of gynecological morbid conditions. It consists in the injection of a radiopaque viscous liquid into the cervical canal and in following the course of the injected material by fluoroscopy through the uterus and the fallopian tubes into the peritoneal cavity. Films are taken to permit more leisurely study of the struc-

tures visualized and to form a permanent record of the procedure.

The advantages of hysterosalpingography are: (1) It will confirm a presumptive diagnosis of intrauterine or intracervical tumor. (2) It permits a detailed radiological exploration of the fallopian tubes. (3) It may act as a therapeutic agent in opening non-patent tubes in cases of sterility from this cause. (4) It permits differential diagnosis between a tumor of the cervix, corpus, or tube, and an adnexal tumor. (5) It makes possible diagnosis of certain conditions which are impossible to diagnose preoperatively without this method, such as (*a*) asymptomatic carcinoma of the endocervix or corpus, (*b*) intrauterine tumors in a uterus of normal size and contour, and (*c*) certain cases of hydrosalpinx.

Contraindications to the use of this procedure are signs of infection and pregnancy.

Twenty roentgenograms; 4 photographs

H. J. THOMPSON, JR., M.D.
Jefferson Medical College

Spina Bifida Occulta and Nulliparous Prolapse (With Notes on the Incidence of Certain Abnormalities of the Sacrum). Arthur G. Gemmell, P. H. Whitaker, and R. L. Plackett. J. Obst. & Gynaec. Brit. Emp. **55:** 459–463, August 1948.

This paper is designed to answer the question: "Is there any relationship between spina bifida occulta and nulliparous prolapse?" (spina bifida occulta being used here to mean non-closure of the posterior arches of the sacral vertebrae). The answer, as derived from the authors' observations on a group of cases collected from their colleagues throughout the British Isles and a control group of representative healthy nulliparous women, is "no," for no statistically significant difference in incidence was found in the two groups. The incidence of sacral anomalies in the normal series was 5.9 per cent, but others have found it to be as high as 25 per cent.

The final conclusion is that "this examination of the present knowledge of the incidence of sacral deformity shows that our information is incomplete. It emphasizes the necessity for the fullest description of the material examined and for the elimination of as many variables as possible. Only when accurate knowledge is available of the incidence of symptomless bony anomalies in healthy individuals will the proper assessment of their role in the cause of symptoms become possible."

S. F. THOMAS, M.D.
Palo Alto, Calif.

Salpingosigmoidal Fistula. Morton Vesell. West J. Surg. **56:** 478–479, September 1948.

The author reports a case of salpingosigmoidal fistula discovered by chance during the performance of hysterosalpingography for sterility. The patient had undergone no operative or obstetrical procedure, nor was there any history of pelvic disease or inflammation. Six months later, when hysterosalpingography was again done, the fistula had healed spontaneously.

Six hysterosalpingograms.

THE GENITO-URINARY SYSTEM

Significance of Calcareous Tuberculous Glands in the Abdomen in Relation to the Urinary Tract. James A. Ross. Brit. J. Urol. **20:** 109–113, September 1948.

Five thousand reports from the Urological Diagnostic Theatre of the Edinburgh Royal Infirmary were reviewed to ascertain the frequency and symptomatology of calcified nodes in relation to the urinary tract. In most cases there had been a full investigation, with retrograde pyelography; in the remainder scout films had been taken, so that in every instance the pelvis and the abdomen had been examined radiologically. All opacities, *e.g.*, gallstones, calcified arteries, phleboliths, as well as calcified nodes and renal calculi, were recorded.

Of the 5,000 cases, 608 (12.16 per cent) showed calcareous nodes. The number and size varied considerably: in some a single node was described; in others the nodes were multiple and widely scattered throughout the abdomen. They were noted in the pelvis, overlying the sacrum, over the sacroiliac joints, the right or left iliac fossa, the line of the attachment of the mesentery, and in the renal regions. The commonest site was the right iliac fossa. In only 25 cases could the nodes be considered a possible cause of symptoms, and in only 10 could they be regarded as actually affecting the urinary tract. These effects consisted of a deviation of the ureter, a bend or kink in the neighborhood of the nodes, with or without slight dilatation of the ureter or pelvis above it, and with or without symptoms. The symptoms consisted of slight pain in the side or mild renal colic. No stenosis of the ureter was found.

It is concluded that unless a definite kink or deviation of the ureter can be shown, the presence of calcareous nodes discovered during the course of an investigation can be regarded as of little significance.

Five roentgenograms; 1 table.

On Tomography as an Adjunct to Urography. Preben Thestrup Andersen. Acta radiol. **30:** 225–236, Sept. 30, 1948.

The author reports 100 cases in which tomography of the kidney was utilized as an adjunct to routine urographic examination. He believes that the procednre is valuable especially in acute cases where preliminary bowel preparation is contraindicated and in outpatients and others in whom adequate cleansing is difficult or impossible. It is useful, also, in the presence of prostatic hypertrophy, which is often associated with marked flatulence.

In cases where gas and fecal material have completely obscured the renal outline and the pyelographic shadow in routine studies, satisfactory visualization has been accomplished on a tomogram taken at a depth of between 5 and 10 cm. The paper is illustrated with reproductions of 4 tomograms and corresponding pyelographic studies.

Some illustrative cases are presented.

P. B. LOCKHART, M.D.
Indiana University

Improved Pyelographic Results in Uretero-Intestinal Anastomosis. Morris Schnittman. J. Urol. **60:** 421–434, September 1948.

An ideal procedure for uretero-intestinal anastomosis has not been perfected, although some sixty technics have been proposed. The criteria of an effective procedure are: no leakage at the anastomosis, normal kidney function, no hydronephrosis, and no complications or mortality. With previous technics, about 25 per cent of postoperative pyelograms were normal, but the usual result was not good. In the series of 11 cases reported

here 81 per cent of the postoperative pyelograms were normal.

The author believes observation of the following principles was responsible for the improvement in his series: mobility of the rectosigmoid, freeing of a minimum of ureter, formation of an adequate trough in the bowel for the ureteral anastomosis, and avoidance of excessive or redundant ureter between its exit from the posterior peritoneum and entrance into the trough.

Twenty-six roentgenograms; 14 drawings of operative technic; 1 table. ALLAN K. BRINEY, M.D.
University of Pennsylvania

Management of the Injured Kidney: Preliminary Report. Kenneth M. Lynch, Jr. J. Urol. **60:** 371–380, September 1948.

A statistical analysis of 23 cases of renal injury is presented. Surgical intervention *versus* conservative management is discussed briefly, with the conclusion that early exploration is advisable wherever a flat film, excretory urography, or retrograde pyelography indicates the likelihood of rupture. The author believes the ideal time for exploration is about seventy-two hours after injury unless shock or severe hemorrhage compel earlier operation.

The primary symptom of renal injury is pain. It was present in 91 per cent of 13 cases classified as ruptures and in 66 per cent of 9 contusions. The principal signs are hematuria, tenderness, rigidity, a mass in the flank, and shock. Frequently there are associated injuries which must assume pre-eminence in the management.

The great importance of roentgenography is pointed out. It is the only method by which rupture can be definitely diagnosed. Flat films were obtained in all but 4 cases of this series. In all 7 cases which were subsequently operated upon the psoas muscle was obscured or obliterated, and this is regarded as evidence of extrarenal hemorrhage unless there is a history of previous renal disease. In 15 of the 23 cases intravenous urography was done. In 2 of these there was no function on the injured side; in 7 filling was poor; in 6 it was good. In 2 cases with good filling there was marked extravasation of the dye. Retrograde pyelography was employed in only 3 cases, in 2 of which there was extravasation of the dye. Urologists differ in their attitude toward this last type of examination in the presence of renal injury, many feeling that it is likely to provoke further bleeding, though no evidence in support of this view is found in the literature.

The treatment depends on the amount of damage found and ranges from simple suture to nephrectomy. Successful use of oxycellulose gauze is described in the report of 1 case. The chief late sequelae are secondary hemorrhage, extravasation of urine, infection, hydronephrosis, calculus formation, calcified cysts, and complete or partial loss of kidney function due to cicatrization.

Five roentgenograms; 2 drawings.
N. F. ZIMMERMAN, M.D.
University of Pennsylvania

Sudden Death Following Intravenous Administration of "Diodrast." Samuel Simon. J.A.M.A. **138:** 127–128, Sept. 11, 1948.

The basis for this report is a fatality subsequent to the intravenous administration of iodopyracet (diodrast) in a young man who had been involved in an automobile accident, following which he had noticed that the urine was blood-tinged. He gave no history of previous allergic manifestations or of any serious illnesses or operations. Preliminary testing for sensitivity to the drug was apparently not done. Death occurred twenty minutes after the injection of 25 c.c. of a 35 per cent aqueous solution, done slowly over a period of five minutes. Three minutes after completion of the injection, the patient vomited, felt numb, and showed a thready pulse and cyanosis. Epinephrine, oxygen, and nikethamide were of no avail.

The literature on the subject is well reviewed, and precautions to be observed in the use of diodrast are outlined. ZAC F. ENDRESS, M.D.
Pontiac, Mich.

An Unusual Variant of Duplication of the Ureter. James A. Ross. Brit. J. Urol. **20:** 125–126, September 1948.

Unilateral duplication of the ureter is frequently encountered. In the majority of these cases there is a small upper pelvis draining approximately one-third of the kidney, with a large lower pelvis serving the remainder. Cases have been observed in which the two divisions were equal in size, but only one case in which the upper pelvis was the larger. Another instance of this latter variety is recorded.

Four roentgenograms.

Primary Benign Neoplasm of the Ureter. Joseph M. Edelstein and Saul M. Marcus. J. Urol. **60:** 409–417, September 1948.

The authors report a case of a primary benign ureteral transitional-cell papilloma, bringing to 32 the number of microscopically proved primary benign ureteral neoplasms reported in the literature. Pain, hematuria, and a palpably enlarged kidney are prominent features of such cases. The pain may be severe and colicky due to the passage of blood clots, or may be dull and aching due to a hydronephrosis. The most prominent and earliest symptom is hematuria, which may be profuse, spontaneous, and intermittent, gross or microscopic. The ureteral tumor itself is rarely palpable. The diagnosis of ureteral tumor depends upon cystoscopy and x-ray examination as well as the history and physical examination. Obstruction to the passage of a ureteral catheter is a suggestive finding. As conditions requiring differentiation on the ureterogram the author lists "malignancy, tuberculosis, inflammatory strictures and calculi," but the differential features are not discussed.

The treatment of ureteral tumor has been total ureteronephrectomy because of the potential malignancy of benign transitional-cell neoplasm, which is the usual type. A diagnosis of carcinoma should be reserved for those cases in which microscopic study shows invasion of the stalk or cells with typically malignant characteristics. It is important, however, to study many microscopic sections in these cases.

Three illustrations, including 1 roentgenogram.
PAUL W. HOFFERT, M.D.
University of Pennsylvania

Unusual Ureterograms in a Case of Periarteritis Nodosa. Russell S. Fisher and Herbert H. Howard. J. Urol. **60:** 398–404, September 1948.

A case of periarteritis nodosa is reported in which in-

travenous urography demonstrated extensive and unusual involvement of the ureters. An intravenous urogram obtained one month after the patient was first seen showed excellent filling of both pelves. The sixty-minute film revealed considerable retention of the dye in both kidneys. The upper thirds of both ureters were markedly spastic and moderately dilated. A similar picture was obtained a month later.

Autopsy revealed marked vascular congestion with numerous hemorrhagic foci throughout both kidneys. The renal pelves were injected but free of exudate. The ureters were not dilated. The walls, especially throughout each upper third, were thickened and edematous. Microscopically there were edema of the ureteral epithelium and a severe panarteritis and arteriolitis throughout the circular muscular wall, and especially in the adventitial connective tissue.

The authors' review of the literature failed to reveal ureterograms such as were found in this case either in periarteritis nodosa or in other pathological conditions. Because the urinary tract is involved in 80 per cent of cases of periarteritis nodosa, it is suggested that urologic examination should be a routine preoperative procedure in cases having bizarre abdominal pain possibly due to that disease.

Four illustrations, including 1 roentgenogram.

JOHN F. WEIGEN, M.D.
University of Pennsylvania

Carcinoma of the Bladder with Bone Metastases: A Report of Eight Cases. Herman L. Kretschmer and J. H. McDonald. Surg., Gynec. & Obst. **87:** 328–337, September 1948.

The authors believe that metastasis to bone from carcinoma of the bladder is much more common than is generally suspected. A review of the literature reveals 105 cases in which this has been known to occur. Males are more often affected than females, most of the cases in both sexes falling in the fifth and sixth decades of life. In the cases in which the duration of urinary symptoms was recorded, an average of twenty-three months elapsed before evidence of bone involvement appeared. Not uncommonly, however, the presenting complaint is pain due to the skeletal metastases. The authors report a series of 8 cases. The tumors varied from small benign-appearing papillomas to large ulcerating masses, the size apparently not influencing the time of the appearance of the secondary bone spread. Dissemination is most generally believed to be via the blood stream, but when the lesion appears in the symphysis pubis or in the ischium, it may be due to direct extension.

Brief histories of the authors' 8 patients are presented with autopsy findings in one and biopsy of the bone lesions in another. In all but one, osteolytic lesions were demonstrated by x-ray. The most common location was in the bones of the pelvis and spine. Lesions were also demonstrated in the ribs, femur, tibia, and clavicle.

Eight roentgenograms; 8 photomicrographs; 2 tables.

ROY GREENING, M.D.
University of Pennsylvania

A Pathologically Displaced Upper Femoral Epiphysis as a Foreign Body in the Urinary Bladder. E. Sadek. Brit. J. Urol. **20:** 114–116, September 1948.

This case of a pathologically displaced upper femoral epiphysis appearing as a foreign body in the urinary bladder is considered to be unique. A 14-year-old Egyptian boy fell and received a superficial laceration on the lower anterior aspect of the left thigh above the level of the patella. This became septic and was dressed for about forty-five days. About five weeks following the injury, the temperature became elevated, and a week later an abscess of the right arm was incised. The fever continued and during the patient's stay in bed it was noticed that both thighs were flexed on the abdomen and attempted extension was painful. The right hip then gradually extended, without any special treatment, but the left thigh continued flexed. Approximately nine months after the injury an x-ray examination of the hip was made and a plaster spica applied. Fifteen days later urinary symptoms developed, but no investigation was made at that time. The plaster spica was replaced in three months and again in six months. As early as two months after the injury it had been noticed that there was pain accompanied by a slight swelling in the buttock over the site of the left sacroiliac joint. Two years after the injury the swelling became inflamed and opened spontaneously, with a discharge of thick pus without any noticeable odor; this gradually became thinner and the sinus closed in about five months. It was about this time the patient came under the observation of the author, with a limp, limited movement of the left knee and hip, and the above-mentioned sinus. He also complained of painful frequent micturition and a sudden stabbing pain in the pelvis on sitting, with a desire to urinate at once. Examination of the urinary tract disclosed a movable foreign body in the bladder. This proved to be the head of the femur. It was removed through a suprapubic incision, and the patient made an uninterrupted recovery.

The author believes that the separation of the femoral head was pathological and not traumatic, because of the absence of any local or general reaction after the manipulation and the comparatively short time (fifteen days) between the manipulation and the appearance of bladder symptoms.

One roentgenogram; 1 photograph.

Polymorphous Cell Sarcoma of the Bladder: Case Report. Victor C. Laughlin, Claire C. Althoff, and Henry W. Brown. J. Urol. **60:** 459–462, September 1948.

The authors place the total number of reported cases of sarcoma of the bladder at 156. To this number they add another. This occurred in a 57-year-old white female complaining of hematuria, frequency, tenesmus, and dysuria. Four months previously a red mass resembling granulation tissue had been removed from the urethra, with a diagnosis of urethral caruncle. Cystoscopy revealed a large bladder with atrophic mucosa; the ureteral orifices could not be seen because of a tumor of considerable dimensions. Pieces of tissue were removed for examination and while the patient was under the anesthetic a pelvic examination was carried out, revealing lateral extension of the mass. X-ray examination, including cystograms and intravenous pyelograms, did not visualize the tumor clearly but the floor of the bladder was shown to be elevated by a soft-tissue structure of increased density as compared with the surrounding structures. The kidney calices, pelves, and ureters were of normal appearance.

Microscopic examination of the removed tissue showed a polymorphous-cell sarcoma. Deep x-ray

therapy (900 r to each of three fields) was followed by rapid diminution in the size of the mass. Three months later both ureteral orifices were stenosed. Dye from an intravenous urogram appeared in both kidneys and apparently they were considered normal. Cystoscopy revealed what grossly appeared to be scar tissue in the wall of the bladder.

Examination one and a half years after removal of the original urethral lesion showed no recurrence of the bladder tumor and no evidence of metastases.

Two photomicrographs. ROY GREENING, M.D.
University of Pennsylvania

Congenital Valvular Formations in the Urethra. Sigvard Jorup and Sven Roland Kjellberg. Acta radiol. **30:** 197–208, Sept. 30, 1948.

Congenital valvular formations in the urethra are relatively uncommon. They usually appear in the posterior urethra as folds or duplications of mucosa, causing varying degrees of obstruction to the flow of urine. Three types are described.

The first type is characterized by a prominent fold running downward from the verumontanum, dividing into two "thin fins" that are fixed to the urethral walls peripherally but are more or less free centrally. Occasionally there is only one such fin. In the second type the fold runs upwards from the verumontanum and then branches into two thin fins that are fixed just below the internal urethral orifice. The third type has the appearance of a diaphragm, extending across the lumen.

The roentgen picture is typical. A urethrocystogram obtained with an opaque water-soluble contrast medium gives the best results. The valves are usually seen best on the micturition films.

In order to prevent further damage to the urinary system over and above that occurring in intra-uterine life, early diagnosis and treatment are essential.

Fourteen roentgenograms; 2 photographs; 4 diagrams. O. R. RUSSELL, M.D.
Indiana University

THE ADRENALS

Nonhormonal Adrenal Cortical Carcinoma: Report of Case with 5 Year Survival and Relief of Hypertension. Zachary R. Cottler. J. Urol. **60:** 363–370 September 1948.

There are few reports of non-hormone-producing tumors of the adrenal. This may be ascribed to: (1) their rarity, (2) the absence of early symptomatology, (3) the possibility that in the late stages of the disease, with metastases, confusion may arise as to the origin of the primary growth.

The non-hormonal type of carcinoma is usually discovered in its late stages, when it is manifested by a palpable mass, vague abdominal and flank pain, and symptoms referable to metastases. The duration of symptoms is from eight months to two years with an average of one year. These neoplasms are seen almost invariably in adults over thirty, without sex predilection.

An important diagnostic aid in the study of such lesions is roentgenography after perirenal air insufflation. This procedure should be used in combination with excretory and retrograde urography and laminagraphy.

A case in a 50-year-old Russian Jewess with complaints referable to pelvic relaxation is presented. Routine abdominal examination revealed the presence of a large hard mass which filled the right side of the abdomen and flank. The blood pressure ranged between 290/170 and 178/140. The remainder of the clinical and laboratory examinations, including hormone assays, were essentially non-contributory. Roentgen studies showed findings consistent with a right renal tumor, but no perirenal air insufflation was done. At operation the right kidney and a large tumorous adrenal were removed. The pathological diagnosis was adenocarcinoma of the adrenal. Following operation a course of roentgen therapy was given to the operative site and abdomen.

Thirty-three months after operation there was clinical evidence of metastasis in the abdomen, and thirty-nine months postoperatively there was radiographic evidence of a superior mediastinal mass. X-ray therapy to the chest and abdomen resulted in no demonstrable change in these lesions, but five years after operation the patient continued in good condition, fairly comfortable, ambulatory, and able to perform her household duties. This is the longest reported survival in a patient with this type of tumor.

Postoperatively the blood pressure returned to normal and remained so. The author believes this is the result of removing an ischemic kidney caused by torsion of the renal pedicle incident to pressure from the adjacent tumor.

Four roentgenograms; 1 photograph; 1 photomicrograph. DANIEL TALLEY, III, M.D.
University of Pennsylvania

HYDATID DISEASE

Hydatid Disease and Its Roentgen Picture. Pablo M. Schlanger and Henriette Schlanger. Am. J. Roentgenol. **60:** 331–347, September 1948.

A review of 470 cases of echinococcosis accentuates the already recognized fact that this disease produces an extremely variable clinical picture, depending on the localization, vitality, and evolution of the parasite, the local complications of infection and rupture, and the presence of metastatic cysts in other parts of the body. These cysts, when occurring in the liver, may rupture or may remain unruptured. In the latter case, they may become calcified. They may become multiple but never grow larger after they once calcify.

The authors describe and illustrate with roentgenograms echinococcus cysts of the abdomen, spleen, kidney, bones, and lung. No statistics are given as to the incidence of the lesions in the various portions of the body.

Forty-eight roentgenograms.
STANLEY H. MACHT, M.D.
Baltimore (Md.) City Hospitals

TECHNIC

A Method of Checking the Centring of X-Ray Tubes. A. D. O'Connor and L. F. Lamerton. Brit. J. Radiol. **21:** 470–471, September 1948.

The exact collimation of the beam is of great importance when small fields are used in roentgen therapy. This can be easily checked by inserting in the proximal end of the cone a lead shield, in the exact center of which is a pinhole. Crossed wires are placed at the distal end of the cone with the intersection at the exact center. A pinhole radiograph will then accurately locate the

focal spot and indicate the correctness of the collimation.

Three illustrations. SYDNEY J. HAWLEY, M.D.
Seattle, Wash.

A Viscous, Water-Soluble Contrast Preparation. Preliminary Report. O. Morales and H. Heiwinkel. Acta radiol. **30:** 257–266, Sept., 30, 1948.

The authors present a preliminary report on a highly viscous non-irritating water-soluble contrast preparation with which they hope to replace iodized oils. The preparation consists of 35 per cent umbradil, 2.5 per cent carboxymethylcellulose (CMC), and 0.25 per cent xylocain. The CMC imparts viscosity to the preparation. The percentage concentration can be altered to vary the viscosity and contrastability to suit the examiner.

Twenty-seven roentgenograms.

J. G. LORMAN, M.D.
Indiana University

About Depth Perception in Viewing Roentgenograms. M. Hopf. Radiol. clin. **18:** 298–301, September 1948. (In German)

Every roentgenogram (excepting tomograms, kymograms, and stereoscopic films) is a central projection of the object. The perspective, therefore, is accurately reproduced. Theoretically and practically this perspective, that is depth perception, can be seen by one eye if it is at the same distance. If one looks at a roentgenogram which is transverse (90 degrees to its normal axis) with one eye for a few minutes, there is a definite plastic visualization; the effect is similar when the roentgenogram is looked at upside down by one eye. A chest film becomes stereoscopic when looked at from a distance of six feet with one eye closed. It seems that we are so used to looking at routine roentgenograms in the normal position of the film on a plane, that it is necessary to shift the axis of the film. A simple apparatus is described for monocular examination of a film in transverse position where the distance between the eye and film can be changed according to the original focus-film distance.

H. W. HEFKE, M.D.
Milwaukee, Wisc.

Illustrated Roentgenograms—A Pedagogic Aid. Louis Shattuck Baer. California Med. **69:** 216–217, September 1948.

The author describes briefly and illustrates effectively a method for clarifying, in terms of three dimensions, the heart shadow on the roentgen film. The technic consists of making preliminary sketches on Klear Pak superimposed on the x-ray film in the proper position After a satisfactory sketch is obtained, the permanent drawing is made with pen and india ink on the x-ray film itself, which is then photographed.

Four illustrations.

RADIOTHERAPY

Irradiation of Pituitary Tumors. Results in Fifty Cases. H. Dabney Kerr. Am. J. Roentgenol. **60:** 348–358, September 1948.

The treatment of pituitary tumors was formerly primarily surgical. Today the principle of irradiation first, to be followed by operation in case of failure, is generally accepted. The author's technic differs from that most commonly employed in that he tries to deliver to the tumor in one course all the radiation that is necessary to cause regression. He uses 200 kv., 1.95 mm. Cu half-value layer, 50 cm. distance, and a 5-cm. cone, treating four fields—two temporal, one frontal, and one vertical. A dose of 100 r is given to one field the first day. If no reaction occurs, two fields are given 100 r each on the second day, after which the dose is increased to 200 r to each of two fields daily until each field has received 2,000 r in air (average tumor dose 2,400 r).

A series of 50 cases is reported, including the following types of tumor: acidophilic, 11 cases; basophilic, 1; chromophobic, 37; mixed acidophilic and chromophobic, 1. Seventy-two per cent of the acidophilic lesions and 70 per cent of the chromophobic showed excellent or good results. The basophilic case did poorly. The age range of the patients was from eleven to seventy years; 26 were females.

The author believes that all types of uncomplicated pituitary tumors should receive initial irradiation except where cyst formation, hemorrhage into the tumor, or increased intracranial pressure is suspected. Visual fields should be checked during treatment and regularly for two months after irradiation. Constriction of the visual fields may be seen during early treatment; when it occurs later, surgical intervention is indicated.

Radiation necrosis of the skull was not seen in this series. Mental deterioration, when it occurred, bore no direct relation to treatment, but on the whole psychotic patients and those with cerebral symptoms did not do well and should probably not receive irradiation. Some autopsied cases revealed no change in the pituitary tumor which could be interpreted as a radiation effect. Why a tumor that has been irradiated and obviously influenced by the treatment (since the patients were symptom-free over a long period) should still show no microscopic evidence of response is difficult to understand. Follow-up of the author's cases which were treated at least six years ago showed no instance of excellent or good result in which retrogression later occurred, and in one case reported as showing a poor result at ninety-six months the condition was excellent at one hundred and sixty-one months.

One table. HARRY J. PERLBERG, JR., M.D.
Baltimore (Md.) City Hospitals

Testosterone Propionate in Treatment of Recurrent Cancer of the Breast. Arthur B. McGraw. Arch. Surg. **57:** 385–390, September 1948.

McGraw reports 12 cases of advanced recurrent breast cancer treated by testosterone propionate. Individual doses were mostly 150 mg., although only 50 mg. were used initially; the aggregate dose varied from 450 mg. to 6,225 mg. Too abrupt initial dosage led to undesirable reactions in the first 2 patients treated; slight masculinizing effects, mostly of the voice, took place in 3 patients, but not to a degree sufficient to create a problem. Six of the patients are dead, but only one failed to show material improvement; in 6, improvement was striking, in 2 good, and in 3 fair. Increased well-being and power to carry on ordinary activities, with improvement or retardation of metastases, were seen in 7, and

reversal of the bone changes took place in 2. The use of this agent as a palliative measure is encouraging, but as an adjunct to, not a substitute for, roentgen therapy.

One table, summarizing the data for the 12 cases.

LEWIS G. JACOBS, M.D.
Oakland, Calif.

The Role of Radiation in the Treatment of Cancer of the Breast. J. Maisin. J. de radiol. et d'électrol. **29:** 363–402, 1948. (In French)

The author, who is Director of the Institute of Cancer, Louvain, Belgium, has made a statistical study of the role of radiation therapy in the management of breast cancer. This involves the experience of the Institute of Cancer of Louvain as well as statistics gleaned from world-wide centers. The statistical conclusions are those which are familiar to American radiotherapists, namely, that irradiation in conjunction with radical surgery improves the five-year survival rate in cancer of the breast associated with axillary lymphadenopathy.

Of more interest for the American radiologist, who is prone to be unfamiliar with the French literature, is the introduction to this extensive work, which beautifully summarizes the experimental biological studies of cancer of the breast and the relation these studies may have to present and future treatment by irradiation. It is believed that a translation of this introduction will be of interest in that it is thought-provoking and opens up new vistas for the possible management of this disease in the future.

"Our knowledge of cancer of the breast has made considerable progress in the course of the last several years.

"The role of hormone imbalance in the etiology and evolution of this type of cancer has been particularly studied. These studies have been made in diverse species of animals, notably in the mouse, the rat, and the rabbit, but the mouse particularly has furnished the most important experimental material. The results obtained are in general agreement for all species studied. It is for this reason that it is interesting to view it in relation to the human clinical material and attempt to derive from it useful conclusions in radiation therapy.

"It is necessary at the outset to state that most of these experiments have been made with homozygous strains of known heredity. The role played by heredity in the determination of results cannot be ignored. In man it is evident that we never have an homogamous condition, the latter having for all time been lost by intermixing. Thus in man we find a condition which is polyzygotous. If, because of this, the question is more complex, the facts established in animals remain nevertheless true, and all these findings have some applicability to man from the biologic point of view.

"The influence of the estrogenic hormones has been particularly well studied by Loeb, Lacassagne, Murray, Little, Bittner, Gardner, Strong, Andervont, Korteweg, and many other workers. It has been established in an irrefutable manner that in strains of homozygous mice predisposed to cancer of the breast the estrogenic hormones play a very certain role in the genesis of this type of cancer. Given in excess, it lowers the age incidence of cancer or augments this incidence, and is likewise capable of causing the appearance of cancer in male mice, which normally do not exhibit it. In many of these strains, if one castrates the females at an early age, the cancer does not appear, if one grafts the testicles from the brother within the strain, the result is the same, and if one administers testosterone, the cancer does not appear.

"These various authors are not in agreement relative to behavior in strains which are not hereditarily disposed and in which large quantities of hormones are applied. Certain workers contend that one is thus able to incite cancer of the breast; others claim that this is impossible.

"In the rat, certain authors, as for example Geschickter, claim that with adequate doses one can provoke a cancer of the breast in animals not formerly predisposed. This question has recently been reviewed by Dunning, Curtis, and Segaloff. These authors have found that even though all strains which have been tested with large and continuous doses of diethylstilbestrol given by implantation have formed cancer of the breast, there are nevertheless important differences in receptivity with varying strains. In fact, certain strains developed cancer in the female breast up to 78 per cent and as high as 68 per cent in the male, while other strains exhibited a complete absence of cancer of the breast. These latter, however, form calculi of the renal pelvis and bladder.

"It is thus certain that some strains of rats and mice, if not all, are sensitive to an excess of estrogenic substances and that the breast is one of the first organs to suffer and eventually to succumb to cancer under the influence of such a physiologic disturbance. This noxious influence of the estrogens is counterbalanced by that of the androgens. We may also glean from these experiments that it is those strains particularly predisposed to cancer which are most sensitive to the estrogens; women who exhibit cancer of the breast probably fall into this category.

"These are, however, not all of the noteworthy observations in animals which seem to have great value in human application.

"Females belonging to certain strains of mice which are castrated shortly after birth likewise form cancer of the breast (Wooley). Likewise it has been shown that these animals exhibit estrus despite castration: the suprarenals secrete estrogenic hormones by a sort of vicarious function following ablation of the ovaries. It is not impossible that in certain women likewise castrated, the adrenals are able to take over the place of the ovary in secreting certain ovarian hormones or hormones which have properties closely allied with those of the estrogens.

"Biskind as well as Gardner and Li have been able to show that under the influence of a prolonged hormonal imbalance one can provoke cancer in the mouse and the rat. Young male rats are castrated and then receive a graft into the spleen of the ovaries of young females. Under the influence of castration the anterior lobe of the pituitary enters into a state of hyperfunction and secretes an abundance of gonadotropic hormones. This latter abnormally excites the ovarian graft in the rat and causes its hypertrophy. Normally the ovarian secretion resulting therefrom should inhibit the gonadotropic secretion of the pituitary. The ovary, however, being nourished in the spleen secretes its hormonal product into the portal circulation where it is destroyed by the liver. It thus never arrives in the general circulation and never reaches the anterior lobe of the pituitary, which thus remains abnormally hyperactive and continues to induce the functional hyperplasia of the ovary within the splenic pulp. This hyperplasia eventually undergoes transformation to neoplasia of a follicular type.

"In certain women at menopause, and in castrated women, it seems logical to think that a comparable hyperplasia of the anterior lobe of the pituitary intervenes, with all its consequences, abnormal secretion of gonadotropic or adrenal hormones with hyperplasia of the adrenals. One might thus understand why beneficial effects of ovarian castration in the premenopausal woman are only transitory.

"On the other hand, hypophysectomy in the mouse retards or suppresses the appearance of cancer of the breast among female mice predisposed to the disease (Korteweg and Thomas).

"These various laboratory experiments merit considerable study by the radiation therapist and the clinician. Perhaps there will one day open to the radiotherapist the new field of functional radiotherapy, that of the adrenal and hypophysis in relation to cancer of the breast.

"But if the laboratory has brought us a series of new facts of possible aid to the therapist in an effort to deal with one of the most frequent and malignant of all forms of cancer appearing in the female, clinical observation likewise brings arguments which might lead us to revise our therapeutic conceptions in this domain. Certain only of these are cited.

"It is abundantly demonstrated that in many cancers of the breast of Stage I (Steinthal) in which careful microscopic study does not show the presence of any cancer in the lymph nodes, death occurs from distant metastases a considerable time after surgery. This demonstrates that the most extensive operation, perhaps disproportionate even in relation to the disease, is, in a number of cases, insufficient to eradicate the cancer.

"It is likewise evident that certain cancers of the breast recur one or several decades after radical surgery. These may be recurrences of the same histologic type as that of the primary tumor. During all this long interval the organism has harbored dormant cancer cells. During all this long period the patient has been in otherwise good health and unlimited in activity. This would seem to prove a certain physiologic mechanism of defense against cancer which may be extremely efficacious. This likewise indicates that it is not always necessary to completely rid the organism of the last cancerous cell in order that the patient, over a period of many years, may carry on in otherwise good health without any clinical sign of recurrent disease.

"It is nevertheless evident that a great many cancers of the breast, like those of the prostate, metastasize to the bones with a surprising frequency. This likewise should indicate to us that the mechanism of these metastases is not entirely governed by physical laws; there likewise exists a physiology in the dissemination of metastases.

"We have attempted hereby to recall certain experimental and clinical observations which should cause one to believe that certain of our therapeutic conceptions need revision, that we need to think of these newer developments in making a new base for our therapy. The radiotherapist, better than anyone, can profit thereby in his treatment. These developments may lead to modifications of technic and perhaps even to totally new methods of management to the benefit of patients in the future."

Forty-six tables and charts.

SIMEON T. CANTRIL, M.D.
Seattle, Washington

Cancer of the Cervix Uteri: A Review of 296 Cases, 1935–1944. John F. Hynes. Am. J. Roentgenol. **60:** 368–381, September 1948.

An analysis of 296 cases of cervical cancer registered over a ten-year period is presented. Both white and Negro patients are included, and all stages of the disease are represented. For the final analysis of results, 88 "indeterminate cases," including recurrent cancer, untreated cases, cases seen in consultation and treated elsewhere, and deaths from intercurrent disease, are omitted.

The author believes that surgical treatment is rarely indicated. He has found a combined radium and roentgen therapy approach to be more efficacious than either alone. The good results obtained in the series reported are attributed to strict adherence to the following principles:

(1) Roentgen therapy should precede radium therapy in most cases.
(2) Whenever possible, treatment should be continuous, being given within six to eight weeks. An interval of more than four weeks between roentgen and radium therapy is hazardous.
(3) The initial treatment plan should be to give the maximum dose tolerated rather than the minimum curative dose.
(4) Unnecessary irradiation of normal tissues should be avoided.
(5) Roentgen treatment should be limited to areas of known and probable disease.
(6) The whole patient requires treatment during and after irradiation.

Roentgen irradiation is given through four pelvic fields, each receiving a total dose (in air) of 2,400 to 3,000 r, two anterior fields being alternated with two posterior on successive days. The factors are: 200 kv., 0.5 mm. Cu filter, 0.9 mm. Cu half-value layer, 70 cm. distance (increased to 100 cm. for patients whose sagittal diameter exceeds 20 cm.). Treatment is given on five or six days a week and is completed in four to six weeks. The beam is not angulated.

Radium therapy is begun after an interval of ten days. A 2:1 tandem 5 cm. in length is used, containing 75 mg. of radium with 1.0 mm. Pt filtration, so arranged that 50 mg. lie in the cervical canal and 25 mg. within the corpus. The usual dose is 4,500 to 6,000 mg. hr.

The five-year results of treatment for 156 cases treated between 1935 and 1942 were as follows:

	Stage I	Stage II	Stage III	Stage IV
Total cases	16	60	57	23
Alive	14	40	24	2
Survival rate	87.5%	67%	42%	9%

The total five-year survival rate for this group was 51 per cent. Of the entire determinate series (208 cases) 57 per cent survived more than three years.

There were five deaths attributable to irradiation. Among the complications of treatment were colitis, severe in 6 cases; late irradiation ulceration of the bladder, 2 cases; vesicovaginal fistula, 3 cases; pyometra 35 cases, 80 per cent of which were in the three-year survival group. One patient out of 20 is estimated to have marked cutaneous atrophy and telangiectasis from the external radiation.

An unbiased comparison of irradiation and surgery is

presented with pertinent remarks as to the difficulty of any such comparison. The author believes that the survival rate in this series is equal to or higher than any reported surgical series, and that radiation therapy is suitable for a larger proportion of patients.

One roentgenogram; 1 anatomic sketch; 14 tables.

HARRY J. PERLBERG, JR., M.D.
Baltimore (Md.) City Hospitals

Cancer of the Cervix Uteri. A Study of Five to Eleven Year End Results. William E. Howes. Am. J. Roentgenol. **60:** 389–402, September 1948.

A review of 230 cases of proved carcinoma of the cervix admitted to the Brooklyn Cancer Institute from 1936 to 1941 is presented. Treatment was varied, including hysterectomy (7 cases), radium and roentgen therapy.

Ordinarily radium was administered after the method used at the Radium Institute of Paris; 4,800 mg. hr. with radium sources in the vagina and uterus was accepted as a therapeutic dose. For 94 women receiving this or a larger dose the survival rate was 25 per cent. For 34 receiving a smaller dose the rate was 17 per cent, but some of this group had received treatment elsewhere as well.

The author's method for determining the ideal location of the radium sources relative to the tumor and routes of dissemination is presented and illustrated in detail by charts and radiographs. The fixed points thus defined allow an approximation of the radium dosage in gamma roentgens.

Complications incidental to radium applications were those referable to the intestinal tract and to the urinary system. Vesicovaginal and rectovaginal fistulas were thought to be due to neoplastic extension rather than radium therapy. On the other hand, a few cases of intestinal and ureteral stricture were attributed primarily to radiation effect. Emphasis is laid upon ureteral stenosis and its sequelae subsequent to radium therapy. [The recent work of Diehl and Hundley (Surg., Gynec. & Obst. **87:** 705–715, December 1948) reveals neoplastic extension to the ureters to have occurred frequently in more advanced cases, as determined by pre-irradiation urinary studies. They further show that non-excessive doses together with packing the uterus (which displaces the ureter approximately 5 to 8 cm. from the cervix) practically eliminates post-irradiation ureteral stenosis as an entity.—H. J. P.]

Roentgen therapy was administered through various systems of portals and each is discussed. Photographs illustrate the usual type of skin reaction and diagrams show depth dose factors. The author's present system is designed to deliver 4,500 r to each parametrium by giving 2,400 to 3,000 r (air) to each of four pelvic portals, two anterior and two posterior, protecting the mid-line structures by a 2-cm. lead strip; all patients are treated in the Trendelenburg position to dislodge ileal loops from the pelvis. After one-third to one-half of the proposed total x-ray dosage has been administered, radium therapy is instituted. A few days later, the remaining roentgen therapy is given. Intravaginal roentgen therapy was used with radium in many cases and instead of radium in selected cases.

The author concludes that "the larger percentage of survivors appears to have been obtained when radium dosage is delivered up to 5.5 erythema doses into the region of the paracervical triangle (parametrium). This is augmented by additional roentgen irradiation approximating 7 erythema doses."

Sixteen illustrations, including 7 roentgenograms; 2 tables.

HARRY J. PERLBERG, JR., M.D.
Baltimore (Md.) City Hospitals

Five Year End-Results of Irradiation Therapy of Cancer of the Cervix Uteri at the Memorial Hospital. Equinn W. Munnell and Alexander Brunschwig. Surg., Gynec. & Obst. **87:** 343–348, September 1948.

This report covers the results in 1,072 patients with cancer of the cervix seen from 1934 to 1941 as submitted from Memorial Hospital (New York) for publication by the League of Nations Health Organization. The over-all five-year cure rate for 1,037 patients treated by irradiation was 28.6 per cent. The cure rate did not show any significant improvement in the period covered.

A divided dose radiation technic was shown to be superior to the massive dose technic employed earlier, but in general radiation therapy was believed to have reached a stage beyond which it is not advancing. It is suggested that possibly surgical attack should be combined with radiation therapy.

Eight tables.

JOHN O. LAFFERTY, M.D.
University of Pennsylvania

An Evaluation of Adjunctive Radiotherapy in the Surgical Treatment of Endometrial Carcinoma. Harold Speert and Thomas C. Peightal. Am. J. Obst. & Gynec. **56:** 502–508, September 1948.

The authors analyzed 157 surgically treated cases of carcinoma of the endometrium at the Roosevelt Hospital. Their analysis shows that there is no benefit from preoperative irradiation with intrauterine radium. In early cases with tumors of low histologic grade the best results were obtained by hysterectomy alone. Postoperative x-ray therapy appeared to be of value in advanced cases and possibly for tumors of high histologic grade.

Seven tables.

JOHN DECARLO, JR., M.D.
Jefferson Medical College

Diagnosis and Treatment of Primary Ovarian Carcinoma with Special Reference to Radiation Therapy. John H. Freed and Eugene P. Pendergrass. Cancer Research **8:** 361–370, August 1948.

The records of 87 patients with a histologic diagnosis of cancer of the ovary who received radiation therapy at the Hospital of the University of Pennsylvania between 1930 and 1941 were reviewed with particular reference to the value of the treatment.

Seventy-two per cent of the patients were from forty to sixty-nine years of age. The incidence of sterility (42 per cent) in the married women in this series was high in comparison to a normal incidence of 10 per cent. This is in accord with the findings of others, suggesting a possible relationship between ovarian cancer susceptibility and sterility.

Pain, abdominal swelling, irregularity of menses, and postmenopausal bleeding were the most frequent presenting symptoms. Ascites was present in 34 patients at operation. A pleural effusion was observed in 8 patients at some time during the course of the disease. Since the pleural effusion disappeared after removal of the primary tumor in 2 women, a possible similarity to a Meigs' syndrome is suggested in some of these cases.

The clinical stage of the disease appeared to be of

greater significance than the histologic type in the treatment and prognosis in this series.

All of the patients had at least an exploratory operation. Whenever possible, a bilateral salpingo-oöphorectomy and hysterectomy were done. Some of the patients referred for treatment after operation elsewhere had only a unilateral oöphorectomy. All of the 87 patients received postoperative irradiation. Two tumors were found to be inoperable at the first operation but, after a full course of roentgen therapy, were so reduced in size so as to be removable at a second operation. One of these patients, with widespread peritoneal implants at the first operation, showed complete regression of the metastatic lesions upon reoperation, after receiving a tumor dose of 1,500 tissue r to the abdomen and pelvis. The second patient showed marked regression of the tumor after receiving a tumor dose of 1,500 tissue r to the pelvis, but the abdomen was not irradiated and the peritoneal transplants were still present. A third patient was found to be inoperable at the first operation and at a second exploratory operation two weeks after receiving 1,000 tissue r to the abdomen and 2,000 tissue r to the pelvis. Two months later, she was again operated on and the tumor growths were completely removed. No metastases were present at the third operation.

In 2 patients in whom only one ovary was removed a carcinoma developed in the opposite ovary fourteen and sixteen months later.

The radiation factors were 160 to 200 kv. (constant potential), 5 to 15 ma., half-value-layer 0.95 to 1.1 mm. Cu, 50 to 80 cm. distance. The pelvis was irradiated through 2 anterior and 2 posterior 15 × 15-cm. or 17 × 17-cm. portals until 1941, after which a single anterior and posterior port, 15 × 20 cm., was used. Cases with evidence of widespread peritoneal transplants were given radiation over the upper abdomen through single 20 × 20-cm. anterior and posterior portals. Four patients with pleural effusion who were thought to have metastatic lesions in the chest were given palliative treatment over this region, using an anterior and a posterior portal, 15 × 15 cm. Patients with a large localized recurrence in the pelvis were given additional treatment over this area. Two patients with a localized lesion in the cul-de-sac received treatment through intravaginal portals in addition to the external irradiation.

With the use of two anterior and posterior portals, a dose of 200 r (measured in air) was delivered to each of two portals daily or on alternate days, depending upon the tolerance of the patient. Occasionally, when the patient lived some distance from the hospital, 3 or 4 portals were treated with 200 r (in air) twice weekly. When single large anterior and posterior portals were used, one portal was given 200 r (in air) daily. The cyclic method was used on 10 patients, treating 2 portals daily for eight to ten days, with a rest period of from one to two weeks between cycles. There was no evidence that this method was producing any better results and it was therefore abandoned in favor of the serial method. Most patients received from 1,500 to 3,000 r (in air) at each series with a rest period of from one to four months or longer between series. Those who were in good general condition and tolerated treatment well were sometimes given a full course of therapy in a single series. In some cases the treatment was spread over two, three, or four series.

Fifty-five patients were given abdominal irradiation as well, with doses varying from 200 to 2,500 tissue r. Of the patients with abdominal carcinomatosis at operation, 14 received less than 500 tissue r and only 2 of these survived more than two years, 4 received from 500 to 1,000 tissue r, 17 were given from 1,000 to 2,500 tissue r.

For purposes of analyzing results, the cases were classified into two groups: (*a*) those receiving a small tumor dose, less than 1,500 tissue r (31 patients) and (*b*) those receiving a larger tumor dose of 1,500 tissue r or more (53 patients). A comparison of the survival rate curves shows a significantly greater number of five-year survivals (47.2 per cent) in the group of patients receiving over 1,500 r (25.8 per cent in the group receiving less than 1,500 r). Three Stage III patients are not included, as complete follow-up was not available. If the Stage I cases, in which many of the five-year cures may be due to surgery alone, are omitted, there remain 63 patients, of whom 25 received less than 1,500 tissue r, while 38 received 1,500 tissue r or more. The five-year survival rate in the group receiving the larger tumor dose is now 36.5 per cent, or 20.5 per cent greater than that for the group receiving less than 1,500 tissue r. This again seems to indicate that radiation increased the number of five-year cures. However, since the percentage difference in the five-year cure rate is not changed significantly by omitting the patients in Stage I, this would suggest that radiation did not have any real effect on the five-year cure rate, *i.e.*, in preventing recurrence, in patients whose tumor was localized to the ovaries and completely removed. The small number of patients in the series makes it impossible to draw any definite conclusions.

A delay of six weeks after operation before starting roentgen therapy is advocated to give the patient a better chance to recover from the surgical procedure.

Eight tables; 3 graphs.

Treatment of Metastatic Bone Tumors. Magnus I. Smedal and Ferdinand A. Salzman. Wisconsin M. J. **47:** 675–686, July 1948.

The pathology, origin, and end-results of 100 consecutive cases of metastatic bone tumor are discussed. Carcinoma of the breast accounted for 24 per cent of this number. In this group the skeletal lesions were predominantly lytic, with 7 showing mixed lesions on the roentgenogram. The spine was most frequently involved. Bone metastases from breast cancer are fairly radiosensitive, but the results of treatment are only palliative. The radiation effect has been said to be increased by the use of colloidal lead. Estrogens and androgens have also been advocated, but their usefulness has not been properly evaluated.

Carcinoma of the prostate will metastasize to any bone in which there is red marrow. Twenty of the cases in the present series were of prostatic origin. The lumbar spine and pelvis were predominantly involved. The authors do not irradiate bone metastases from prostatic cancer, preferring castration and hormone therapy.

Bone metastases from the kidney, thyroid, and lung are all radioresistant. Relief of pain may be obtained, but the course of the disease is not altered. Radioactive iodine has shown promise in the treatment of metastases from the thyroid. According to those who have had experience with this method, the selective uptake of radioactive iodine depends upon two conditions: first,

the thyroid must be removed or irradiated to make it inactive; second, the uptake in metastases is higher in those which more closely approach normal thyroid structure.

The authors feel that the discrepancy in figures from various clinics for metastatic bone lesions is due to improper roentgen screening of the skeleton. They advise a routine survey in all cases of malignant disease, including a single film of the chest, a study of the thorax, including the shoulders, lateral roentgenograms of the skull and cervical spine, anteroposterior and lateral studies of the thoracic and lumbar spine, and an anteroposterior film of the pelvis and hips.

Fourteen roentgenograms; 9 tables.

LOUIS BERNSTEIN, M.D.
Hartford, Conn.

Roentgen Therapy in Traumatic Myositis Ossificans. Ernst A. Pohle and Carol Tomlinson. Am. J. M. Sc. **215:** 372–380, April 1948.

The process of calcification and ossification occurring in muscle tissue in response to trauma is called traumatic myositis ossificans. The condition tends to become asymptomatic and regress, but it may be disabling and even progressive. It is particularly common on the anterior aspect of the thigh, following a blow, and in the elbow region, following dislocation.

It is generally accepted that the lesion begins as an inflammatory response to hemorrhage and absorption, during which ossification occurs. Acute exudative inflammation, hyperplasia, and organization of connective tissue are the earliest histopathologic findings. Islands of osteoid tissue later become surrounded by osteoblasts and marrow spaces. Cartilage is frequently present.

The most constant symptom is pain of varying duration and persistence. If the lesion is near a joint, limitation of motion may result.

Roentgenographically, calcification may appear from one to four weeks after injury. The bone formation may reach its maximum in anywhere from six weeks to twenty months. Regression may occur in from two months to years after the discovery of the calcification.

The prophylactic measures most widely advocated are those attempting to reduce the hemorrhage immediately after the injury, such as avoidance of massage, and the application of cold and pressure bandages. After calcification has occurred, surgical removal is indicated only if the lesion is near a joint or in the origin or insertion of a muscle.

The technic employed in radiation therapy calls for doses of 150 to 200 r (in air) to one or two fields daily, or every other day, for three or four treatments. When indicated, a second series may be given in four to six weeks, and a third, two to four months after the first course. Technical factors in the series here reported were 175 to 400 kv., 50 cm. focal skin distance, and half-value layers of 1.05 mm. Cu and 2.4 mm. Cu. The field included a wide zone about the area of calcification.

Ten cases were treated, and complete relief of pain occurred in all, in four to six weeks. Increased density and a sharper and smoother outline were noted in all cases, but these changes need not necessarily be due to irradiation. Beneficial results appeared to be definitely related to roentgen treatment. The action of the rays is believed to be similar to that in chronic bursitis and other inflammations.

Since surgical excision is often followed by recurrence, the results obtained by radiation therapy justify further trial even in cases of long duration.

Six roentgenograms; 1 photomicrograph; 1 table.

BENJAMIN COPLEMAN, M.D.
Perth Amboy, N. J.

Treatment of Cancer of the Penis. Bruno Bertiglia. Radiol. med. (Milan) **34:** 540–547, September 1948. (In Italian)

The author had the opportunity of observing 30 examples of cancer of the penis at the Radium Institute of Bologna between 1930 and 1942. He stresses the frequency of congenital phimosis, which was found in 13 of these cases. In all instances a biopsy was done and the lesion proved to be squamous-cell epithelioma. Twenty-three of the 30 patients had inguinal adenopathy. The author stresses, however, that not all inguinal adenopathies were metastatic. He points out that in a series reported by Colby these nodes were found to be inflammatory and not malignant in 14 out of 32 cases.

It is believed that better results can be obtained in penile carcinoma by a combination of surgery and radiation than by radiation alone. Five out of the 7 patients treated by surgery and radiation were still alive at the end of five years, while only 5 of 18 treated by radiation alone survived for a similar period. The author believes that by judicious choice of methods of treatment, one should obtain five-year survivals approaching 40 per cent.

Contact therapy was used in 9 cases (7,000–11,000 r); radium needles were employed in 12 cases, and radium over a plastic mold in 12 cases. Deep x-ray therapy was given to the inguinal regions (1,500–1,800 r in five to six days). In 2 cases a simple amputation was done and in 1 amputation with excision of the lymph nodes. Four patients underwent excision of lymph nodes only and 2 had complete castration. Surgery was always followed by radiation therapy to the operative scar and to the inguinal areas. The author was impressed by the heavy reaction of the corpora cavernosa following irradiation.

CESARE GIANTURCO, M.D.
Urbana, Ill.

Impressions of Developments in Radiology Abroad. R. Kaye Scott. M. J. Australia **2:** 253–258, Sept. 4, 1948.

These scattered remarks on radiotherapy as practised in America and Great Britain do not lend themselves to abstracting. Mention is made of them for the sake of those who may be interested in the impressions made upon a visiting radiologist by a few of our leading institutions. It is of interest that the author advocates the separation of radiodiagnostic and radiotherapeutic training.

RADIOACTIVE ISOTOPES

Biologic Effect of Irradiation by Radioactive Iodine. Bengt N. Skanse. J. Clin. Endocrinol. **8:** 707–716, September 1948.

Studies with radioactive iodine, I^{131}, in doses which have been used as "tracers" were undertaken with two main objectives: (1) to determine the minimal amount of radioactive iodine which may interfere with the normal function of the thyroid and (2) to study the effects of irradiation by radioactive iodine on various known functions of the thyroid. The purpose of the present paper is to report the radiation effect of I^{131} on the thyroid in respect to collection of radioactive iodine, capacity for growth, capacity to collect iodine (from food and water), capacity to respond to thiouracil, and capacity to respond to thyrotropic hormone.

Five-day-old cockerels were injected with 0.5 unit of thyrotropic hormone for three consecutive days. Twenty-four hours after the last injection, three groups were injected subcutaneously with I^{131} (containing 0.1 microgram sodium iodide as a carrier) in doses of 1, 10, and 50 microcuries, respectively. A fourth group received no radioactive iodine and was used as a control.

Determination of thyroid collection curves for 0.5 μc, 1 μc, 10 μc, and 50 μc of I^{131} demonstrated that during the first 96 hours no physiologic change took place in the thyroid at any dose level. The earliest radiation effect was observed in the 50 μc. group 144 hours after the administration of the isotope, indicating that the thyroid does not retain a large dose of I^{131} as well as it does a smaller one for any extended period.

Thyroid of chicks which had received 1 μc. of I^{131} did not change in growth or iodine concentration. In fowls which received 10 μc. and 50 μc. the normal growth of the thyroid was significantly inhibited. Iodine concentration was not altered in chicks which received 10 μc, but there was a significant decrease in concentration of thyroid iodine in the 50 μc group.

All irradiated chicks responded to thiouracil as measured by increase in thyroid weight twenty-six days after receiving I^{131}. However, 38 days after they received I^{131} there was no longer any response to thiouracil in the 50 μc. group, and in the 1 μc. and 10 μc. groups the response was not as marked as in the non-irradiated controls.

All the irradiated chicks responded to thyrotropic hormone as measured by increase in thyroid weight and loss of iodine 16 days after administration of I^{131}. However, at the 24-day interval there was demonstrated a dissociation in response to thyrotropic hormone between the 10 μc. and 50 μc. group. In the 10 μc. group a loss of iodine was observed but no increase in thyroid weight. In the 50 μc. group there was no effect on thyroid weight and the loss of iodine was minimal.

Five charts; 3 tables.

A Study of the Histopathology and Physiologic Function of Thyroid Tumors, Using Radioactive Iodine and Radioautography. Brown M. Dobyns and Beatrice Lennon. J. Clin. Endocrinol. **8:** 732–748, September 1948.

A radioautographic technic was used to study the affinity for iodine, or function, of thyroid adenomas in 94 cases of nodular goiter. The term "adenoma" is used to describe any completely encapsulated discrete tissue mass in which the histologic pattern is different from the remaining thyroid tissue. The rather universal assumption that the degree of affinity for radioiodine represents the degree of function of the thyroid tissue is accepted. The term "hyperfunctioning" is therefore used to describe an adenoma which, by its increased affinity for a tracer dose of iodine, demonstrates the utilization of iodine in excess of the remaining thyroid tissue.

All patients were given an identical standard tracer dose of radioactive iodine (I^{131}) twenty-four to seventy-two hours before surgery. The nodules were removed together with an adjacent piece of thyroid tissue. The block composed of both types of tissue was prepared by a rapid histologic technic, mounted on slides, and exposed to film. Six radioautographs, each with a different exposure time, were made of each adenoma.

It was found that if adenomas are arranged as a spectrum, beginning with the least differentiated or most embryonal and progressing onward to the most differentiated types, in general the degree of differentiation runs parallel to the degree of function.

There are adenomas which have cellular hypertrophy and hyperplasia and which hyperfunction, but there are also adenomas which have cellular hypertrophy and hyperplasia which scarcely function at all. The group with negligible function shows increased variability in the cell height and in this respect appears to shade off into the class of tumors known as papillary adenocarcinoma.

Adenomas which are functioning in excess of the remaining thyroid tissue may occur with or without an elevation in the basal metabolic rate. By their action they apparently suppress the function of the remaining thyroid tissue, depending on the magnitude of their contribution to the total biologic demand for thyroid hormone.

Twenty-five illustrations, including 8 radioautographs.

Dosimetric and Protective Considerations for Radioactive Iodine. James J. Nickson. J. Clin. Endocrinol. **8:** 721–731, September 1948.

The desirability of knowing as accurately as possible the amount of radiation received by the thyroid is discussed, together with the uncertainties associated with the calculations employed. The need for determining as soon as possible the amounts of I^{131} which are carcinogenic or otherwise toxic to the normal organism and the importance of the current and future clinical work in the resolution of the present uncertainty are stressed. Constant awareness of the toxic properties of radioactive materials by those conducting investigative or diagnostic studies in human beings is of great importance.

The need for protection of the worker against radiations and radioactive materials is discussed and protective measures are outlined.

Radioactivity and Urinary-Tract Calculi. David S. Cristol, Albert E. Bothe, and Paul W. Grotzinger. New England J. Med. **239:** 427–429, Sept. 16, 1948.

This interesting case report concerns a 56-year-old male in whom a 3 × 4-cm. calculus was found in the urinary bladder. Numerous prostatic calculi were also

present. The patient also had a polycythemia vera, for which radioactive phosphorus was given. He received 6 millicuries by mouth and five months later an additional 10 millicuries by mouth. Throughout this period he was observed frequently and the bladder calculus was seen to grow. Sixty-one days after the last dose of radioactive phosphorus, the bladder calculus and many of the prostatic calculi were removed.

Examination of the bladder calculus with the Geiger counter, showed a radioactivity of 2,000 counts per minute at a distance of 10 cm. Autoradiography of the vesical stones also showed very definitely a ring of radioactivity around the calculus; there was no evidence of radioactivity in the central portion. The prostatic calculi showed no radioactivity.

These findings point to a new way of investigating the development of urinary calculi and will probably be of some value in the study of this problem.

One roentgenogram; 2 autoradiographs; 1 photograph.

JOHN B. MCANENY, M.D.
Johnstown, Penna.

EFFECTS OF IRRADIATION

The Hazards of X-Ray. Editorial. J. A. M. A. **138:** 214–215, Sept. 18, 1948.

Attention is called to this editorial because of its reemphasis on the dangers inherent in the use of x-rays. The principal points it makes are as follows:

Roentgen rays for diagnosis (films and fluoroscopy) are safe only because radiologists have a long tradition of being careful and because films and screens are so sensitive as to require rather small exposures. In treating cancer, resulting damage to skin and normal structures is severe and obvious, but such damage, including the possibilty of late roentgen ulceration, or late roentgen cancer, is not too high a price to pay for cure of a cancer. When benign conditions are concerned, the point of view is different. Roentgen treatment for such cases should be used only with a vivid appreciation of its capacity for harm and with an overt evaluation of its presumptive benefits weighed against the known and possible injuries inseparable from its use in effective dosage.

Mortality of Medical Specialists, 1938–1942. Louis I. Dublin and Mortimer Spiegelman. J. A. M. A. **137:** 1519–1524, Aug. 21, 1948.

This study is based on a tabulation of full-time medical specialists listed in the American Medical Directory of 1940 and the deaths among corresponding specialists within the five-year period from 1938 to 1942. The data relate only to physicians residing within the United States. Of the 37,010 specialists in the United States, 1,595 were radiologists and roentgenologists (0.9 per cent of all physicians; 4.3 per cent of all specialists).

With regard to physicians specializing in roentgenology and radiology, the authors make the following statement: "Although the mortality ratio for all causes for roentgenologists and radiologists, namely, 90 per cent, is below that for non-specialists, it lies well above the average for all specialists. In the case of coronary disease and cancer, their mortality ratios, 110 per cent and 133 per cent, respectively, exceed those for the non-specialists. The high mortality from cancer among roentgenologists and radiologists may be of special significance when viewed in conjunction with their mortality from leukemia. Five of the 95 deaths among roentgenologists and radiologists in this experience were from leukemia; no other specialty had as many or as high proportion of deaths from this cause. The five deaths due to leukemia recorded for roentgenologists and radiologists is several times the number expected on the basis of the mortality experience of all male physicians, the standard of comparison. Although roentgenologists and radiologists constituted only 4.3 per cent of the living specialists, they had one quarter of the deaths from leukemia among all specialists. The facts are indicative of an extra hazard to roentgenologists and radiologists that may arise from their exposure to dangerous radiation."

Radiation Hazards in Industry. Charles R. Williams. J. Indust. Hyg. & Toxicol. **30:** 294–299, September 1948.

With the advent of nuclear energy and increasing production of radioisotopes for research and industrial use, radiation as an industrial hazard has aroused widespread interest. The author discusses some of the applications of radiations (excluding infra-red and ultraviolet) in industry and the problems associated with them. Under the heading X-Ray he takes up installations of 400 kv. and less, high-voltage (1,000,000 and above) equipment, shoe-fitting fluoroscopes, anti-theft and anti-sabotage fluoroscopes, high-vacuum electronic tubes, and under Radioactive Substances radiography, linear sources, nickel-polonium alloys in spark plugs, the radium-type vacuum gage, self-luminous paints, and the use of thorium in the manufacture of gas mantles.

Protection Against Radiation Hazards and Maximum Allowable Exposure Values. Karl Z. Morgan. J. Indust. Hyg. & Toxicol. **30:** 286–293, September 1948.

The author describes the measures taken at the Oak Ridge National Laboratory to protect workers from radiation damage and discusses the maximum allowable exposure values. He states that the tolerance level of 0.1 r per day chosen five years ago for the Plutonium Projects is not as low as it should be. Health physicists consider 0.1 r per day as the maximum permissible dose of radiation and about 0.01 r per day as the permissible operating dose. [The National Committee on Radiation Safety has recently reduced the "permissible dose" to 0.3 r per week.] A recent analysis of the personnel radiation exposure records kept by Health Physics personnel monitoring sections (Oak Ridge) indicates that no employee is averaging more than 0.02 r. per day.

Four photographs; 2 tables.

Complications Following Irradiation of the Thyroid Gland. R. M. Lukens. Ann. Otol., Rhin., & Laryng. **57:** 633–642, September 1948.

X-ray irradiation of the larynx for carcinoma is justified in cases of inoperable cancer and as after-treatment in cases in which remaining concealed cancer cells are suspected. However, when x-ray is resorted to in treatment of other conditions in close proximity to the larynx and trachea (notably lesions of the thyroid gland), consideration must be given to the possibility of damage to the underlying larynx and trachea.

Cartilages of the larynx and trachea are susceptible to intensive irradiation. The degenerative process is slow and may not give alarming symptoms until several years later. The damage is permanent, progressive, and requires a lifetime of treatment. In addition, the patient is constantly in danger of death due to asphyxia. In the cases herein reported, obstruction to the airway was due not only to pathologic narrowing of the larynx and trachea but also to altered secretion accumulating at the point of stenosis. One patient lost her life because crusts became wedged in the narrowed airway during the night and help could not be obtained quickly enough to save her.

Symptoms exhibited in the 5 cases recorded were hoarseness, sensation of lump in the throat, inspiratory dyspnea, wheezing, dysphagia, cough, expectoration, loss of weight, pain in the chest. The physical findings were congestion of the laryngeal mucosa, telangiectasis and scarring of the neck, stenosis of the larynx, inflammation of the laryngeal mucosa, telangiectasis of the vocal cords, stenosis of the trachea, granulomatous lesions in the tracheal walls, crowding inward of the tracheal wall, viscid adherent tracheal secretions, crusting. Cicatrices are not common; only a suggestion of cicatricial tissue was present in one case.

The time between termination of the irradiation and appearance of the first symptoms of tracheal disease was one year in 2 cases, three years in 2 cases, and seven years in 1 case.

Tracheotomy was required in 3 of the reported cases. This may be rendered hazardous by changes in the blood vessels in the overlying tissues and lowered resistance to infection. There may be delayed healing, with necrosis and gangrene of the operative field in some instances.

No statement is made as to the dosages of radiation employed in the cases presented.

STEPHEN N. TAGER, M.D.
Danville, Ill.

Contributions to the Study of Pleuropulmonary Modifications Accompanying Radiation Therapy of Breast Cancer. R. Sarasin, G. Voluter, and J. Garcia-Calderon. J. de radiol. et d'électrol. **29:** 445–452, 1948. (In French)

The authors have reviewed the literature relative to radiation fibrosis of the lung and have presented a number of their own observations on this complication. Their conclusions are as follows:

The reaction of the lung to x-rays is always an exudative one. This exudation involves both the pleura and the parenchyma. A considerable atelectasis is generally associated with this mechanism. An inflammatory component has also been found, the origin of which is not always irritative. Exacerbation of specific pulmonary disease or non-specific inflammation may be associated with reaction in the mediastinal lymph nodes and even in the contralateral lung and pleura. All of these reactions may be transitory. They likewise, however, predispose the tissue for the later appearance of fibrosis and are the first step leading to an eventual post-roentgen fibrosis of the lung and pleura.

The forms which this exudative and atelectatic process may take are varied. There may be phases of regression or progression and occasionally these may end in a total disappearance of the process. The end-result may be a generalized emphysema, a discrete acinous sclerosis, pleural adhesions, or finally parenchymal fibrosis, the degree of which may be extremely varied.

Twelve roentgenograms.

SIMEON T. CANTRIL, M.D.
Seattle, Wash.

On a Peculiar Late Reaction in Radiologically Treated Cases of Cancer of the Hypopharynx. Preliminary Observations. Sölve Welin. Acta radiol. **30:** 249–256, Sept. 30, 1948.

Of 327 patients treated for histologically verified cancer of the hypopharynx, 195 were followed by repeated roentgen examinations. Of these, 5 patients exhibited a picture which was strongly suggestive of a local recurrence, showing increased width of the prevertebral soft-tissue shadow and irregularity of the mucosa in the area of the former tumor. On direct and indirect hypopharyngoscopy, however, no evidence of recurrence could be demonstrated. The changes appeared as early as three months after irradiation and as late as eleven months. They disappeared without further treatment. The original treatment was the same in these 5 cases as in the remainder of the series, approximately 5,600 to 6,000 r delivered to the tumor in thirty days.

The changes are believed to represent a late irradiation reaction, though the author suggests the alternative possibility of some extrinsic factor such as infection.

Fourteen roentgenograms. J. SCOTT, M.D.
Indiana University

Radiation Myelitis of the Cervical Spinal Cord. Geoffrey Boden. Brit. J. Radiol. **21:** 464–469, September 1948.

Myelitis of the cervical cord secondary to irradiation for various lesions in the mouth, pharynx, and neck was found in 10 of 161 cases treated between 1942 and 1946. Four cases were transient and six progressive.

The symptoms appeared between one and fifteen months after irradiation; in half of the cases the period was more than nine months. Subjective symptoms were the only indications in the transient cases. These included numbness and tingling of the neck, shoulders, and arms. In the progressive cases the symptoms went on to paresthesias, weakness or paralysis in one or more limbs, and in the late stages bowel or bladder dysfunction. The physical signs in the progressive cases were those of transection of the cervical cord. Five patients died.

Four patients had been treated through multiple small fields, and in 3 of these progressive myelitis developed. The dosages had been, respectively, approximately 2,000 r to the cord in one day; 2,225 r in one day; and 5,200 r in seventeen days. One patient who received approximately 5,060 r in seventeen days had only transient myelitis.

Six patients had been treated with large fields which included the whole neck and the full length of the cervical cord. Three patients who received less than 3,375 r in seventeen days showed progressive symptoms.

The author concludes that long lengths of the cord should not receive more than 3,500 r in seventeen days. Patients given 5,000 r in seventeen days or 2,000 r in one day are apt to show symptoms of transverse myelitis.

Three tables. SYDNEY J. HAWLEY, M.D.
Seattle, Wash.

Cytologic Changes in Thymic Glands Exposed in Vivo to X-Rays. Robert Schrek. Am. J. Path. **24:** 1055–1066, September 1948.

In previous work Schrek found that x-rays killed thymic cells (lymphocytes) *in vitro* after a latent period of three hours or more. During this latent period primary intranuclear vacuoles developed in many of the cells. After twenty-four hours of incubation, nearly all of the irradiated cells were dead and these cells then showed small, round, single, dark, partly encapsulated, secondary vacuoles. These same changes occurred in non-irradiated suspensions incubated at 37° C. but at a slower rate than in irradiated suspensions. It was thus concluded that x-rays did not initiate any new degenerative process but accelerated the normal aging and death of the lymphocyte.

In the study recorded here an attempt was made to determine whether or not similar phenomena occurred in thymic tissue *in vivo*. Anesthetized rats and rabbits were treated with 1,000 r over the thymic gland and were sacrificed one to six hours later. Counts were made of the dead cells and of the live cells of portions of the glands and estimates of the percentage of viable cells were thus determined. Dark-field study for the presence of primary and secondary vacuoles was done.

Intranuclear, acidophilic vacuoles were produced in many cells three hours after irradiation. Many of the irradiated cells had single or multiple primary vacuoles with or without vacuolar walls. Five hours after irradiation many cells showed pyknotic fragmented nuclei. Many of the cells of this tissue had secondary vacuoles. Controls (non-irradiated thymus) also showed a few such changes. The spontaneous x-ray-induced degeneration of lymphocytes was associated with the formation of intranuclear vacuoles and fragmentation.

Seven photomicrographs.

STANLEY H. MACHT, M.D.
Baltimore (Md.) City Hospitals

Comparative Examinations about the Influence of X-rays and Chemical Substances on Mitosis. K. Hohl. Radiol. clin. **18:** 302–309, September 1948. (In German)

Mitoses are relatively easily disturbed by extrinsic factors—radiation as well as chemical compounds. With radiation the primary effect, that is absorption of quanta, is essentially the same by protons, neutrons, roentgen rays, and ultraviolet rays. Experiments on mitosis of root meristems of plants show that the first changes are observed in about thirty minutes in the early metaphase of the mitosis. In forty-five minutes to an hour definite changes are also seen in the later metaphase, evidenced by pyknosis. Dosages of 25 and 50 r in air were not effective as far as visible changes were concerned; but dosages of 100 r (in air) produced definite pyknosis. The effect seems proportionally larger when 300 r are delivered. Only trypaflavine seems to have a similar pyknotic effect, without damaging other plant structures.

Summation of irradiation and chemical effects takes place when dosages of each are used, which are by themselves not harmful. Nitrogen mustard seems to act like radiation on the centromer. Stilbamidin had no effect on the mitosis of plants. Other anti-mitotic substances, as urethane, chinone, etc., are discussed.

H. HEFKE, M.D.
Milwaukee, Wis.

August 1949

formation

T, M.D.
Hospitals

fluence of
K. Hohl.
n German)
y extrinsic
ompounds.
sorption of
neutrons,
riments on
at the first
utes in the
ve minutes
n the later
es of 25 and
ble changes
) produced
portionally
rypaflavine
out damag-

Tects takes
ich are by
d seems to
midin had
nti-mitotic
scussed.
ER, M.D.
kee, Wis.